A PROSPECTIVE OPEN LABELED RANDOMIZED CLINICAL TRIAL ON

"VALI AZHAL KEEL VAYU"

WITH

"AKKINI CHOORANAM"

Dissertation Submitted To

THE TAMIL NADU Dr. M.G.R. Medical University

Chennai – 32

For the Partial fulfillment for the Award of Degree of

DOCTOR OF MEDICINE (SIDDHA)

(Branch – I, POTHU MARUTHUVAM)

DEPARTMENT OF POTHU MARUTHUVAM

Government Siddha Medical College

Palayamkottai – 627 002.

OCTOBER – 2018

GOVERNMENT SIDDHA MEDICAL COLLEGE
PALAYAMKOTTAI, TIRUNELVELI-627002,
TAMILNADU, INDIA.

Phone: 0462-2572736 / 2572737/ Fax:0462-2582010
Email: gsmc.palayamkottai@gmail.com

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69, Anna Salai, Guindy, Chennai - 600 032.

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TIRUNELVELI – 627 002
TAMIL NADU INDIA

Ph.No : 0462-2572736 / 2572737 / 2582010 **Fax : 0462-2582010**
Email ID : gsmc.palayamkottai@gmail.com

R.No.GSMC / 5676 / P&D / Res / IEC / 2014 **Date : 20.07.2016**

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Address of Ethical committee	Government Siddha Medical College Palayamkottai - 627002 Tirunelveli District
Principal investigator	Dr.P.Priyanga M.D (s) First Year PG Dept of Pothu Maruthuvam Reg.No :
Supervisor	Dr.A.Manoharan M.D (s) Professor & Head of the Department
Guide	Dr.A.Manoharan M.D (s) Professor & Head of the Department
Dissertation topic	A prospective open labeled randomized clinical trial on **"Vali Azhal Keel Vaayu" (Rheumatoid Arthritis)** with evaluation of trial drug **"Akkini Chooranam"**
Document field	1. Protocol 2. Data Collection Form 3. Patient Information Sheet 4. Consent form
Clinical / Non Clinical trial Protocol	Clinical trial protocol - Yes
Informed consent document	Yes
Any other document	Case sheet, Investigation document
Date of IEC approval & it's Number	GSMC/3-IEC/2016-I-5/20.07.2016

We approve the trial to be conducted in its presented form.

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Certificate of Botanical Authenticity

Certified the following plant drugs used in Siddha formulation (Internal) **"AKKINI CHOORANAM"** for **VALI AZHAL KEEL VAAYU** (RHEUMATOID ARTHRITIS) taken up for Post-Graduation Dissertation Studies by Dr.P.Priyanga PG Scholar MD siddha, Department of Pothu Maruthuvam are correctly identified and authenticated through Visual inspection / Organoleptic Characters / Experience, Education & Training Morphology Microscopically and Taxonomical methods.

Table 1: Ingredientof Akkini Chooranam

S.N	Drugs	Botanical Name	Family	Part Used
01	Seeragam	*Cuminum cyminum*Linn	Apiaceae	Fruits
02	Thippili	*Piper longum.*Linn	Piperaceae	Dried Spike
03	Chukku	*Zingiber officinalis.*Rose	Zingiberaceae	Rhizome
04	Lavangam	*Szygium aromaticum.*Linn	Myrtaceae	Flower bud

Station: Palayamkottai

Date : 6. 2. 2017.

Authorized Signature

Dr. S. SUTHA, M.Sc.,M.Ed.,Ph.D.,
Associate Professor
Dept. of Medicinal Botany
Govt. Siddha Medical College
Palayamkottai, Tirunelveli - 2.

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Date first received	:	12.02.2017
Date received after modification (if any)	:	NA
Date received after second modification (if any)	:	NA
Approval date	:	15.02.2017
Expiry date	:	31.07.2017
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15/2/17

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पूजप्पुरा, तिरुवनंतपुरम, केरल
SIDDHA REGIONAL RESEARCH INSTITUTE
Poojappura, Thiruvananthapuram, Kerala

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(आयुष मंत्रालय, भारत सरकार)
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has participated in the CME Programme held on 13.06.2018 at Conference Hall Special Therapy Wing, Government Siddha Medical College, Palayamkottai, Tirunelveli. This Programme is focussed on

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Prof. Dr. A. MANOHARAN, M.D.(s) Ph.D.,
Head, Department of Pothu Maruthuvam (PG)
Government Siddha Medical College, Palayamkottai.

Prof. Dr. R. NEELAVATHI, M.D.(s) Ph.D.,
PRINCIPAL
Government Siddha Medical College, Palayamkottai.

INTERNATIONAL JOURNAL OF CURRENT RESEARCH IN CHEMISTRY AND PHARMACEUTICAL SCIENCES (IJCRCPS)

ISSN : 2348 - 5213 (PRINT); ISSN : 2348-5221 (ONLINE)

www.ijcrcps.com

IMPACT FACTOR: 6.988; ICV:63.58(2016)

Editorial Board Member of

"International Journal of Current Research in Chemistry and Pharmaceutical Sciences"

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Priyanga P*[1], Seetha Lakshmi T[2], Manoharan A[3]

*1 PG Scholar, Department of Pothu Maruthuvam, Government Siddha Medical College (The Tamilnadu Dr. M.G.R. Medical University, Chennai, Tamil nadu, India), Palayamkottai, Tirunelveli 627002, Tamilnadu, India. drpriyapadmanathan@gmail.com

2 PGScholar, Department of Pothumaruthuvam, Government Siddha Medical College (The Tamilnadu Dr. M.G.R. Medical University, Chennai, Tamilnadu, India), Palayamkottai, Tirunelveli 627002, Tamilnadu, India baboo1726@gmail.com

3 Professor and Head, Department of Pothumaruthuvam, Government Siddha Medical College (The Tamilnadu Dr. M.G.R. Medical University, Chennai, Tamilnadu, India), Palayamkottai, Tirunelveli 627002, Tamil nadu, India drmanoharan25@gmail.com

In recognition of the publication of the paper entitled **"Anti-inflammatory activity of *Akkini Chooranam* – A Siddha preparation"** published in IJCRCPS Journal, Volume: 5, Issue: 7, Year: 2018.

Managing Editor
DARSHAN PUBLISHERS
8/173, Vengayapalayam,
Seerappalli (Po.), Rasipuram (Tk.),
Namakkal (Dt.)-637 406.
Tamil Nadu. India.

Editor in Chief
IJCRCPS
Website: www.ijcrcps.com
E-mail: editorijcrcps@gmail.com

ACKNOWLEDGEMENT

I am extremely grateful to my **Lord Almighty** who empowered me with his blessings and grace to finish my dissertation work successfully.

I express my whole hearted thanks to **Prof. Dr. R. Neelavathy, M.D(s)., Ph.D.,** Principal, Government Siddha Medical College, Palayamkottai for permitting me to use the facilities available in the institution for my dissertation work.

I express my whole hearted thanks and gratitude to Professor **Dr.A.Manoharan, M.D(s)., Ph.D.,** Head of the Department, Pothu Maruthuvam Department, Government Siddha Medical College, Palayamkottai for his valuable guidance in each and every step and encouragement in my dissertation work.

I would take this moment to signify my sincere gratitude to **Dr. T. Komalavalli, M.D(s)., Associate Professor**, **Dr. S. Justus Antony, M.D(s)., Dr. G. Subash Chandran, M.D(s)., Ph.D., Dr. S. Chitra, M.D(s)., Dr. S. Uma Kalyani, M.D(s)., Dr. P. Sathish Kumar, M.D(s).,** Assistant Lecturers in Post Graduate Department of Pothu Maruthuvam for valuable suggestions for my dissertation work.

Also I thanks to **Mrs. S. Sudha M.Sc., Ph.D.,** Associate professor, Department of Medicinal Botany and **Prof. Mrs. N. Nagaprema, M.Sc., M.Phil.,** Lecturer, Head of the Department and other Technical Staffs, Department in Biochemistry, Government Siddha Medical College, Palayamkottai for her suggestion in Technical aspect to this work.

I express my thanks to Librarian **Mrs. T. Poonkodi, M.A., M.L.I.S.,** for permitting me to utilize the college library and **Dr.N.Chidambaranathan, M.Pharm., Ph.D., principal K. M. College of Pharmacy,** Madurai for my dissertation work.

LIST OF ABBREVIATIONS

%	-	Percentage
i.e.,	-	That is
RA	-	Rheumatoid Arthritis
ESR	-	Erythrocyte Sedimentation Rate
ASO	-	Anti-Streptolysin 'O' factor
TJC 28	-	Tender Joints 28
SJC 28	-	Swollen Joints 28
DAS 28	-	Disease Activity Score 28
VAS	-	Visual Analog Scale
gms	-	Grams
kg	-	Kilogram
mg	-	Milligram
dl	-	Decilitre
ml	-	Milli litre
cm	-	Centimeter
S E M	-	Structural Equation Modelling
ANOVA	-	Analysis Of Variance
Hb	-	Haemoglobin
TC	-	Total Count
MCV	-	Mean Corpuscular Volume
DC	-	Differential Count
MCHC	-	Mean Corpuscular Haemolgobin Concentration
P	-	Polymorphs
L	-	Lymphocytes
E	-	Eosinophils
CRP	-	C-Reactive Protein
WBC	-	White Blood Corpuscles
RBC	-	Red Blood Corpuscles
HLA	-	Human Leukocyte Antigen
MHC	-	Multi Histo Compability
ACPA	-	Anti-Citrullinated Peptide Anti bodies
TNF	-	Tumour Necrosis Factor

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ABSTRACT

Vali Azhal Keel Vayu is one of the commonest disease now a days, number of suffers increases day by day. The evidence of the disease **VALI AZHAL KEEL VAYU** was derived from ***"Sabapathy Manuscript of Book Noi Nadal Noi Mudhal Naadal Thirattu Part – II" 2nd Edition, by Dr. M. Shanmugavelu, B.H.I.M, (Page No. 623).*** The signs and symptoms metnioned in the Literature of Siddha Book closely resembles with "Rheumatoid Arthritis" in modern medicine.

Out of 40 patients, 20 In patients and 20 Out patients of both sex were selected. They were administered with the clinical trial medicine **"AKKINI CHOORANAM"** 4.2 gms BID with sugar during the whole study period. Akkini chooranam was chosen for this clinical study with reference from ***"Koshayi Anuboga Vaithiya Bramma Ragasiyam, Part – II, Pg.No.104.***

The clinical trial drug was subjected to Biochemical and Pharmacological anal;ysis. At the end of the Clinical trial study, the majority of the cases which showed good results.

CHAPTER-I

INTRODUCTION

The Siddha medicine was found by Siddhars. Siddhars have a unique and added to supernatural powers.

Siddha medicine is found in south India. It is based on the 96 Udal Thathuvam, including Panjapootham and Naadi.

The three thathuvas are Vatham, Pitham, Kabam.

The Panjapoothams are Mann, Neer, Thee, Vayu and Akayam.

These five Panjapoothas are the basic principles of the siddha system.

According to Sathaga Naadi,

"பாரப்பா பூதமைந்து மண்நீர் தேயு
பரிவாயு வாகாய மைந்தி னாலே,
சேரப்பா சடமாச்சு மண்ணின் கூறு
செறிமயிர்தோல் என்பிறைச்சி நரம்பைந் தாகும்
நேரப்பா அப்புவின்கூ றுதிர மச்சை
நீர்மூளை சுக்கில மோடைந் தாகும்
காரப்பா தேயுக்கூறு பயமாங் காரங்
கடுஞ்சோம்பல் நித்திரைமை துணங்க ளஞ்சே".

"அஞ்சான வாயுவின்கூறு யிருத்த லோடல்
அவைநடத்தல் கிடத்தலுட னிருத்தலஞ்சாம்,
அஞ்சாகு மாகாயக் கூறு காம
அதிற்குரோதம் உலோபமோக மதமஞ் சாகும் "

Relations between the panjapootham and tridosam

Vayu + Akayam = Vatham

Thee = Pitham

Mann + Neer = Kabam

Disturbances of this three dhosa are causes to the disease.

According to Thiruvalluvar,

"மிகினும் குறையினும் நோய் செய்யும் நூலோர்

வளிமுதலா எண்ணிய மூன்று".

Vatham is placed first.

According to Theraiyar,

"வாதமலாது மேனி கெடாது"

According to Yugi vaidhya cinthamani, Vatha disease were classified into 80 types.

Among 80 types of vatha disease, vali azhal keel vayu is one of the type of 10 keel vayu.

The symptoms of vali azhal keel vayu is correlated in modern science is Rheumatoid Arthritis.

In 2005 Rheumatoid Arthritis was estimated to affect 1.3 million adults in the U.S. representing 0.6 percent of the population.

By 2007, an estimated 1.5 million adults had Rheumatoid Arthritis.

A 2010 study found that about one-fourth to one half of all patients with Rheumatoid Arthritis become unable to work within 10 – 20 years of follow –up.

From 2012 the increased rate of hospitalization was found in both sexes, all age groups and throughout disease duration.

In 2015, estimated national indirect costs of Rheumatoid Arthritis related absenteeism from work were 252 million annually.

Siddha system has standard and cost effective treatment for "Vali Azhal Keel Vayu".

So, I have selected "Vali Azhal Keel Vayu" for the clinical study with Akkini chooranam for my dissertation work, on the basis of the Siddha.

CHAPTER–II

AIM AND OBJECTIVE

AIM

Vali Azhal Keel Vayu is a disease cause more pain and disabilities that involves poor as well as rich people.

So, I selected this disease and treated the cases with the help of அக்கினி சூரணம் (**AKKINI CHOORANAM**) 4.2 gms with sugar 2 times morning and night after food internally.

The Objectives are:-

- To evaluate the therapeutic efficacy of **AKKINI CHOORANAM**(internal) in the treatment of **"VALI AZHAL KEEL VAYU"** (Rheumatoid Arthritis).
- To explore the apt definition, Aetiology, clinical features, pathology, diagnosis, prognosis, complications and treatment for Vali Azhal Keel Vayu in Siddha literatures and its correlation with modern science.
- To survey the incidence of the disease, according to Age, Occupation, Socio Economic status, Habits, Family History, Paruva kaalangal, Thinai and Three Vital Humours.
- To divulge how the Mukkutram and Seven Udal kattugal are deranged in this disease.
- To document the changes in the Envagai Thervugal in this disease.
- To do a detailed Clinical investigations.
- To use Modern parameters to confirm the diagnosis and prognosis of the disease.
- To analyse the Clinical trial of the drug in Biochemical and Pharmacological studies.
- To do a safety profile of a clinical trial of the drug "**AKKINI CHOORANAM**".

CHAPTER-III

REVIEW OF LITERATURE

a). SIDDHA ASPECTS

The concepts of Siddha system are based on fundamental principles of five basic elements viz; Panchabootham – Mann, Neer, Thee, Kaatru, Aahayam, 96 Thathuvangal, Three Vital Humours and Seven Udal Kattugal.

The three physical constituents of the human system i.e., the humours are Vatham, Pitham, Kapham. They are called Uyirthatus or Thiri thathus under normal condition, which regulates all the physiological activities of the human body and keeps healthy.

வாதநோய்க்கான இயல்பு

"சந்திரவாத முடம்பு குளிர்ந் தெழுந்தே நடுக்குஞ் சீதாவாய் வாம்
முந்திய குந்தி சிவந்து சந்துகள் தோறுங் குடைந்து மொளிகள் வீங்கும்
வந்திய தொந்த வாதம் நரம்புகளெல்லா மிசிந்து வலம் விடாது
அந்து அவ்வாகு வாதம் வீக்கமுண்டா முடலிற்றி முருண்டாமே"

- தேரையர் வாகடம் பாடல் – 211

Pain in the upper and lower limbs , pain in the costo chondral junction will be seen in vatha disease .

According to Theraiyar,

"பொற்றா மரையான் புனைமெய் யரண்காக்கும்
பொற்றா மரையான் புகல்வதென் பொற்றாம்
வளவினிலே யாக்குரம்பை மன்னென்ன மன்ன
வளவினிலே யாக்கும் வளி"

- தேரையர் யமக வெண்பா.

Vatham is being hailed as the king ,who rules the body and enables the dwelling

of the soul (Uyir) in the body. Hence Theraiyar said, Vatham as the prime force.

“வாதம் வந்துற்ற போது வயிறது பொருமி கொள்ளும்

தாதவிழ்ந்திடுப்பு கைகால் சந்துகள் கடுப்பு தோன்றும்

சீதொரு மலமும் நீருந் சிறுத்துடன் கடுத்து விழு

மாதவமரை மேல் வந்த வாதத்தின் குணமிதாமே”

- ***யூகி முனிவர் பெருநூல் வைத்திய காவியம் 1000***

When vatham is increased, abdominal discomfort, pain in the hip joint and in the joints of upper and lower limbs, decreased and painful voiding of stools and urine will be seen .

“சொல்லவே வாதமது மீறிற்றானால்

சோர்வடைந்து வாயுவினால் தேகமெங்கும்

மெல்லவே கைகால்க சைதியுண்டாம்

மெய்முடங்கும் திமிரவொண்ணாத் திமிரு ண்டாகும்

வல்லவே யுடல் பொருமும் வயிறுளைக்கும்

விரும்பி யன்னச் செல்லாது விந்து நஷ்டம்

கொள்ளவே நாப்புளிக்கும் கழிச்சலுண்டாம்

கூறினார் மலை முனி கூறினாரே”

- ***அகத்தியர் சிகிச்சா இரத்தின தீபம்***

வாதம் மிகும்போது வாயு மிகும். சன்னி தோஷம் போன்ற பல வியாதிகள் வந்து சேரும் .உடல் மெலிவு, உடல் சோர்வு, கை கால் அசதி, உடலில் திமிருண்டாதல், வயிற்றுப் பொருமல் , கழிச்சல் விந்து நஷ்டம் , நாவைப் புளிக்கச் செய்தல் போன்ற குறிகுணங்கள் உண்டாகும்.

According to Theriyar,

“தக்கவாயு கோபித்தால் சந்துவுளைந்து தலைநோவா

மிக்கமூரி கொட்டாவி விட்டங்கெரியு மலங்கட்டும்

ஒக்கநரம்பு தான்முடங்கு முலர்ந்து வாய்நீ ரூறிவரும்”

- தேரையர் வாகடம் (பாடல் எண்.42, பக்கம் எண்.13)

According to Agasthiyar,

"காணப்பா வாதமீறில் கால்கைகள் பொருத்து நோவும்

பூணப்பா குடல் புரட்டும் மலசலம் பொருமிக்கட்டும்"

- அகத்தியர் வைத்திய காவியம் 1500

(பாடல் எண்.10. ப. எண்.2)

According to Thiruvalluvar in Thirukural explains,

"மிகினும் குறையினும் நோய் செய்யும் நூலோர்

வளிமுதலா எண்ணிய மூன்று"

Based on this theory, the problems of various systems are classified as Vatha, Pitha, and Kapha diseases.

According to Thirumoolar,

"எறிய நல்வாதம் எறிக்கும் குணங்கேளு

குறியெனக் கைகால் குளைச்சு விலாச் சந்து

புறியென நொந்துடல் பச்சைப்புண் ஆகுமே"

- திருமூலர் கருக்கிடை வைத்தியம்-600

(பாடல் எண்.36, பக்கம் எண்.11)

KEEL VAYU

சித்த மருத்துவத்தில் எண்பது வகை வளி நோய்கள் பற்றிக் கூறப்பட்டுள்ளது. வளி அழலக்கீல் வாயு என்பது, கீல்வாயு என்ற தலைப்பின் கீழ் கூறப்பட்ட பத்து வகைகளுள் ஒன்று. கீல்வாயு என்பது மூட்டு மற்றும் அதனைச் சுற்றிள்ள பகுதிகளில் ஏறப்டும் நோயக்ளைக் குறிப்பதாகும்.

Vali Azhal Keel Vayu comes under the heading of Vatha diseases,

According to Yugi vaidhya cinthamani,

"என்னவே வாதந்தா னெண்பதாகும்

இகத்திலே மனிதர்களுக் கெய்யுமாறு

பின்னவே பொன்னதனையே சோரஞ்செய்து

பெரியோர்கள் பிராமணரைத் தூடனித்தும்

வன்ன தேவச் சொத்தில் சோரஞ்செய்து

மாதாபிதா குருவை மறந்த பேர்க்கும்

கன்னவே வேதத்தை நிந்தை செய்தால்

காயத்திற் கலந்திடுமே வாதந்தானே''

- ***யூகி வைத்திய சிந்தாமணி, பாடல் 243.***

Frittering away Gold, Blaspheming elders, Holymen and Vedas, Obsession of temple property; Obliviousness of parents, Teachers and God all leads to vatha disease.

வேறு பெயர்கள் (SYNONYMS):

The synonyms in **AGASTHIYAR GUNAVAGADAM** text is,

சந்துவலி, மூட்டுவலி, மேகசூலை, முடக்கு வாயு, ஆமவாதம், சந்து வாதம், சூலைகட்டு, சந்திக சிலேஷ்ம ரோகம், வாதசூலை, வாயுரோகம்.

''தானான கீல்வாத ரோக பேரை

சாற்றுகிறேன் நீயறிய விபரமாக

மானான வாய்வுரோகம் வாத ரோகம்

மகத்தான முடகுவாயு முடக்கு வாதம்

தேனான சந்தீக சிலேட்டும ரோகம்

தெளிவான கைகாலில் பிடிப்பு ரோகம்

ஊனான ரசவாதம் சூலைகக்ட்டு

உத்தமனே சந்திவாதம் வாதசூலை யாமே

ஆமென்ற இத்தனையும் அதறகுப் பேராம்''

- அகத்தியர் குணவாகடம்

காரண பெயர்கள்:

நோய் காரணம்	:	மேக சூலை
முக்குற்ற நிலை மாறுபாடு	:	வாத சூலை, சந்திக சிலேஷ்ம ரோகம்
இடத்தைக் கொண்டு	:	சந்து வாதம், மூட்டுவலி, சந்து வலி, ஆம வாதம்
குறி குணங்களைக் கொண்டு	:	சூலைக்கட்டு, முடக்கு வாதம்

இயல் (DEFINITION):

"வளியு மையுந் தன்னிலை கெட்டு
வலியுடன் வீக்கச் சுரமும் காயந்து
மூட்டுக டோறும் முடுக்கியே நொந்து
மூட்டுக டன்னின் நீரும் சுரந்து
தாங்கொணா வலியுடன் நொந்திடு மம்மே"

- ***சபாபதி கையேடு***

According to **Sabapathy Manuscript:** In Keel Vayu, the following symptoms are produced, that is painful joints, swelling associated with fever, restriction of joint movements, finally produce deformity and systemic illness.

நோய் வரும் வழி (AETIOLOGY):

"வாதமலாது மேனி கெடாது"

- ***தேரையர்***

According to Siddha system, any modifications (or) disturbances in Uyir thathugal, especially in Vatham, is a major role to produce Keel Vayu.

According to Sabapathy manuscript ,

"வளிதரு காய்கி ழங்கு
வரைவிலா தயிலல் கோழை
புளிதயிர் போன்மி குக்கு
முறையிலா வுண்டி கோடல்

குளிர்தரு வளியிற் றேகங்

குனிப்புற வுலவல் பெண்டிர்

களித்தரு மயக்கம் பெற்றோர்

கடிசெயல் கருவியாமால"

-சபாபதி கையேடு- சித்த மருத்துவம் (பொது)

(பக்கம்.எண்.624)

There are various number of factors, that play a major role in modification of Vatham.Excessive intake of rhizomes and vegetables that can increase the risk of Vatha diseases.Irregular food intake, prolonged exposure to cold air, staying in hilly area,excessive sexual activity and hereditary factors produce Keel Vayu.

"ஆடியாதியாய் ஐப்பசி ஈறாய்

ஆனிலமதற் கோரரசியல் காலம்"

- சதகநாடி (நோய் நாடல்)

(பாகம்.1, பக்கம் எண்.167,168)

According to Sathaganaadi, the Vatha diseases are predominant in the months of Aadi to Iypasi (July to November).

"வாத வர்த்தனை காலமேதோ வென்னில்

மருவுகின்ற ஆனி கற்கடகமாகும்

ஆதவைப் பசியோடு கார்த்திகை தன்னில்

அருடமே....."

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.245, பக்கம் எண்.76)

In Yugi Chinthamani, Vatham provokes in its own site in Aani and Aadi (தன்னிலை வளர்ச்சி). But it provokes and spread beyond the site, in the month of Iypasi and Karthigai (வேற்றுநிலை வளர்ச்சி). And reassumes normal in the rest of the months (தன்னிலை அடைதல்).

According to this Poem,

“பதுமத்தைப் பூக்க வைக்கும் பானுமிக்க காயும்

முதுவேனி லிற்பு விநீர் முற்றும் - கதுமென

வற்றும் கபகும் வாயுமிகும்.....”

- மருத்துவர் தனிப்பாடல்

முதுவேனிற் காலத்தில், சூரிய வெப்பத்தின் காரணமாக பெருவாரியாக நீர் ஆவியாக்கப்பட்டு பூமியில் வறட்சி நிலவும். அதுபோல் நமது உடலில் வறட்சி ஏற்பட்டு வளிநோய் வருவதற்கு ஏதுவாகிறது.

In pararasa Sekaram ,

காணவே மிகவுண்டாலும் கருதுபட்டினி விட்டாலும்

மானணை யார்கண் மோகமறக்கினு - மிகுந்திட்டாலும்

ஆணவ மல கடம்மையனே விடாதலாலும்

வானுதன் மடநல்லாளே வாதங்கோபிக்கு காணே- 232

அதிக அளவு உணவு உட்கொள்ளல் (Excessive food intake), பட்டினி கிடத்தல் (Starvation).

According to Agasthiyar in Agasthiyar Gunavagadam,

“தானான கீல்வாத ரோகம் பேரை

.....

போமே தான்ரச தூஷியத் தினாலே

பொல்லாத இந்த நோய் காணும் பாரு”

- அகத்தியர் குணவாகடம்

Keel Vayu occurs due to dietary substances which degrade the quality of chyle (அன்னரசம்– chyle).

In Pararasa Sekaram,

“பாரினிற் பயப்பட்டாலும் பலருடன் கோபித்தாலும்

காரெனக் கருகியோடிக் கழுமரத் துரத்தினாலும்

ஏர்பெறு தனது ரெசின் மிகத்துக்க மடைத்திட்டாலும்

பாரிய காற்றினாலும் படரினும் வாதங்காணும்" - ***பாடல் எண் : 233***

பயம் (Fear) , எல்லோரிடமும் கோபம் கொள்ளல் (Arrogant towards all people), மிகுதியாக ஓடல் (Excessive running), துக்கம் (Sadness), தினமும் உடலின் மேல் காற்று படல்.

According to Theraiyar in Theriyar Vagadam text,

"வெய்யிலில் நடக்கையாலும் மிகத் தண்ணீர் குடிக்கையாலும்

சேய்யிழை மகளிரைச் சேர்ந்தன பவிக்கையாலும்

பையனே உண்மையாலும் பாகற்காய் திண்கையாலும்

தையலே வாதரோகம் சனிக்கு மென்றறிந்து கொள்ள"

தேரையர் வாகடம்

(பாடல் எண்.16, பக்கம் எண்.5)

Excessive exposure in sun rays, excessive intake of water, excessive sexual activity, Excessive intake of bitter guard etc…, may disturb the normal functions of Vatham.

Vatha Kanma Varalaru says,

"நூலென்ற வாதம் வந்த வகைதானேது

துண்மையாய்க் கன்மத்தின் வகையைக் கேளு

காலிலே தோன்றியது கடுப்பதேது

கைகாலில் முழக்கியது வீக்கமது

கோலிலே படுகின்ற விருட்சமான

குழந்தை மரந்தனை வெட்டல் மேல்தோல்சீவல்

நூலிலே சீவஜந்து கால் முறித்தல்

நல்லகொம்பு தழைமுறித்தல் நலித்தல் தானே"

அகத்தியர் கன்மகாண்டம்

(பாடல் எண்.56, பக்கம் எண்.23)

Psychological factors such as removing the bark of living trees, injuring the animals, cutting the branches in the living trees and plucking the leaves may produce Vatha diseases.

According to Yugi Muni in Yugi Vaidhya Chinthamani,

"தானென்ற கசப்போடு துவர்ப்பு கைப்பு
சாதகமாய் மிஞ்சுகினும் சமைத்த வண்ணம்
ஆனென்ற ஆறினது புசித்த லாலும்
ஆகாயத் தேறலது குடித்தலாலும்
பானென்ற பகலுறக்க மிராவிழிப்பு
பட்டினியே மிகவுறுதல் பாரமெய்தல்
தேனென்ற மொழியார் மேற்சிந்தை யாதல்
சீக்கிரமாய வாதமது செனிக்குந்தானே"

- யூகி வைத்திய சிந்தாமணி-800
(பாடல் எண்.244, பக்கம் எண்.76)

Excessive intake of bitter, astringent and salty foods, drinking polluted rain water, irregular sleep pattern, undue starving, excessive weight lifting and sexual perversion can induced Vatha diseases.

According to pararasa sekaram,

"தொழில்பெறு கைப்புக் கார்த்தல் துவர்த்தல் விஞ்சுகினுஞ் சோறும்
பழையதாம் வரகு மற்றைப் பைந்திணை யருந்தி னாலும்
எழில்பெறப் பகலு றங்கி இரவினி லுறங்காத தாலும்
மழைநிகர் குழலி னாளே வாதங்கோ பிக்குங் காணே"

-பரராச சேகரம்

கைப்பு, கார்ப்பு, துவர்ப்புள்ள பதார்த்தங்களை அதிகமாகச் சேர்த்துக் கொள்ளல், பழைய சோறு, வரகு, தினை உண்ணல், பகலில் உறங்கி இரவில் விழித்திருத்தல்.

According to Yugi Muni in Yugi Vaithya Chinthamani,

"பகரவே வாதமது கோபித்தப்போ

பண்பாக பெண்போகம் அதுதான் செய்யில்

நகரவே வெகுதூர வழிநடக்கில்

நளிரான காற்றுமே னிமேல் பட்டால்

மிகரவே காய்கள் கனிகிழங்கு தன்னை

மிகவருந்தி மீறியே தயிர்தான் கொண்டால்

முகரவே முதுகெலும்பை முறுக்கி நொந்து

முழங்காலும், கணுக்காலும் கடுப்புண்டாமே"

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.215, பக்கம் எண்.89)

Excessive sexual activity (or) desire, walking for a long distance, prolonged exposure to cold, harmful consumption like taking excessive curd after eating fruits, vegetables and tubers produces toxic factors which affects bones and muscles, produce Vatha diseases.

As per Karmic Law:

In Siddha system, many diseases are said to be precipitated by Kanmam, which means the deeds good (or) bad committed, by an individual in his / her previous and the present births. According to Agasthiyar Kanma Kandam-300, Vatha diseases may also be precipitated by Kanmam.

"அந்தணர் கற்பு மாதர் அருளிய சாபத்தாலும்

முந்திய வினையாலும் முதிர்கர்ப்ப மேகத்தாலும்

சிந்தையிற் கொடுமையாலும் சிவகுரு நிந்தையாலுந்

தொந்தமாம் வியாதியாலும் தோன்றிடும் சூலைதானே"

- அகத்தியர்

Soolai may occur by the curse of well characterized people and ladies or due to evil deeds in the previous births or due to megam produced by their parents or due to bad thoughts and curse of Guru.

Individual derangements in Mukkutram i.e., Vatham, Pitham and Kapham:

- Abanan and Viyanan are affected in Vatham.
- In Pitham, Sathaga Pitham is affected.
- In Kapham, Santhigam is affected.

Classification of Keel vayu:

According to **Sabapathy manuscript** the Keel vayu is classified into 10 types. They are:

1.வளி கீல் வாயு

2.அழல் கீல் வாயு

3.ஐய கீல் வாயு

4.வளிஅழல் கீல் வாயு

5.வளிஐய கீல் வாயு

6.அழல் வளி கீல் வாயு

7.அழல் ஐய கீல் வாயு

8.ஐய வளி கீல் வாயு

9.ஐய அழல் கீல் வாயு

10.முக்குற்ற கீல் வாயு

General clinical features of keel vayu:

1. Painful joints
2. Swelling
3. Restricted joint movements
4. Stiffness
5. Fever
6. Loss of appetite
7. Synovial effusion

VALI AZHAL KEEL VAYU:

Vali Azhal Keel Vayu is one among the ten types of keel vayus.

When the Vadha dosham is in vitiated condition,some untold activity,food stuffs,kanmam which provoke the Pitha dosham which is the causative factor of Vali Azhal Keel Vayu.

வளி (வாத) வடிவத்தன்மை:

"மண்ணி ரனல்கா லென்னு நான்கும்

நிரலே நாற்றந் தட்பந் திட்பம்

உருவி லூற்ற முடைய வாகி

மெய்ப்பொருள ழிபொரு ளெனவிரு வகைய

அனுக்கள் மெய்பொருள் காரிய மழிபொருள்".

வாதத்தின் இயற்கை வடிவம் நுண்மை(அனுத்துவம்)நொய்மைகளும்:

"விடய மரமுத லசைதற் கேது"

என்பதால் இயக்கமும்

"தேலொன் றானே கவர்குண முறது

தட்பம் வெப்ப மவற்றின் வேறென்

றம்மு வளைகத்தா யாயுங் காலை

நிலநி ரனல்கால் நான்கினு நிலையும்".

என்பதால் தன்மை வெம்மை இவ்விரண்டான் வரும் ஒப்பரவின்மையும்(சருச்சரையும்) ஆகிய வளிவடிவத் தன்மைகளாம்.

வாதத்தின் இயற்கை பண்பு:

ஒழுங்குடன் தாதேழ் முச் சோங்கி இயங்க

எழுச்சிபெற் எப்பணியுமாற்ற-எழுந்திரிய

வேகம் புலன்களுக்கு மேவச் சுறுசுறுப்பு

வாகளிக்கும் மாந்தர்க்கு வாயு.

-(சித்த மருத்துவாங்கச்சுருக்கம்)

The normal structure and functions of **Vatham (வடிவத்தன்மை)** is:

- Dry (வறட்சி)
- Cold (குளிர்ச்சி)
- Rough (கடினம்)
- Motion (அசைதல்)
- Light (இலகு)

The normal structure and functions of **Pitham (வடிவத்தன்மை)** is:

- Heat (வெப்பம்)
- Sharpness (கூர்மை)
- Lubrication (நெய்ப்பு)
- Relaxation (நெகிழ்ச்சி)
- Motion (இயக்கம்)

The normal structure and functions gets modified in Vali Azhal Keel Vayu.

வாத மிகு குணம்:

"அறியவிம் மூன்றின் தன்மை சொன்னார்நந்தி

ஏறிய நல்வாத மெறிக்குங் குணங்கேளு

குறியெனக் கைகால் குளைச்சு விலாசச்ந்து..."

- திருமூலர் கருக்கிடை வைத்தியம் 600

வாதவீறு அன்னமிறங் காது கடுப்புண்டாம் வண்ணமுண்டாம்

மோதுகட்கு ரோகம் சுரமுணட்டா மிருமலுமா முறங்காதென்றும்

ஓதுதரிய வாதமனலாகு நடுக்கமுண்டாம் பொருள் களயர்ந்த

தீதெனவே நரம்பித்து சந்துகள் தோறுங் கடுக்குந் தினமுந்தானே"

- ***தேரையர் வாகடம் (பாடல் எண்.210, பக்கம் எண்.58)***

"தக்க வாயு கோபித்தால் சந்துளைத்து சூலைநோவா

மிக்க கொட்டாவி விட்டங் கெரியு மலங்கெட்டும்

ஒக்க நரம்பு தான் முடங்கு மலர்ந்து வாய் நீரூறிவரும்

மிக்க குளிரும் நடுக்கமாய் மேனி குன்றி வருங்கானே"

- ***தேரையர் வாகடம் (பாடல் எண்.43, பக்கம் எண்.13)***

வாதம் மிகும்போது பசியின்மை, உடல் கடுப்பு, சுரம், இருமல், உறக்கமின்மை, உடல் நடுக்கம், நரம்புத் தளர்ச்சி, சந்துகள் தோறும் குடைதல், விலாச்சந்துகள் நோதல், வயிறு பொருமல், குடலிறைச்சல், மலச்சிக்கல், மிகுந்த கொட்டாவி போன்ற குறி குணங்கள் தோன்றும்.

பித்தம் மிகு குணம்:

கண், மலம், சிறுநீர், தோல் இவைகள் மஞ்சள் நிறம் அடைதல், பசி, நீர்வேட்கை மிகுதிப்படல், உடல் முற்றும் எரிச்சல் உண்டாதல், குறைந்த தூக்கம் போன்ற குறி குணங்கள் தோன்றும்.

So, In Vali Azhal Keel Vayu, Vatha Pitha dosham, it produce stiffness, swelling, restriction of movements in the affected joints.

தானான கீல்வாத ரோகம் பேரை

.....

பொல்லாத இந்த நோய் காணும் பாரு

நாமேதான் முழங்கால் பெரியகீல்கள்

நன்மையுடன் அதைச் சுற்றி இருக்கும் சவ்வின்

....."

- அகத்தியர் குணவாகடம்

According to Agasthiyar Gunavagadam, Vali azhal keel vayu affects all joints and their periarticular surface. So, the Vali Azhal Keel Vayu is affecting major, minor joints and end stage it can produce the disability.

Prodromal symptoms of Keel Vayu (முற்குறி குணங்கள்):

Nasal block, running nose, hoarseness of voice, low grade fever, painful arthralgia are common symptoms of Vali azhal Keel vayu.

CLINICAL FEATURES OF VALI AZHAL KEEL VAYU:

In pararasa Sekaram ,

சொர்சீதே வுதிரவாத சுரோணித முழங்கால் தானும்

பொற்கணைக் காலுந் சந்தும் புறவடி தானும் வீங்கி

நற்கணு விரல்க னொந்து நடுப்பயித்திய வாதத்தில்

உற்பவக் குணமுண்டா முறுநூலிற் சொன்னதாமே

- Swelling of ankles and knee joint

- Swelling of hind foot

- Pain in the interphalangeal joints

- characters of paithya vatham are seen

According to Sabapathy Manuscript,

"வாத பித்தக் கீல் வாயுவின்

வருங்குறிச் சாற்றக் கேளாய்

ஏதமார் மந்த மேப்பம்

இரைச்சலும் வயிற்றிற் காணும்

ஓதருங் குத்தல் வீக்கம்

ஓய்தலில் எரிச்ச லுண்டாம்

காதறு முறக்க மின்மை

காய்ச்சலுங் காணுங் கண்டாய்"

Sapapathy manuscript

Vali azhal keel vayu have the symptom of indigestion (i.e) Mantham,Aeppam, Eraichal (Borborygmus of the abdomen . This disease occur due to excessive intake of foods which increases vatha and pitha (e.g, mutton egg, fish potato) and frequent intake of liquers .Laziness may also be produce this disease.

In this disease first eruction due to indigestion then the formation of gas in the abdomen, constipation and obesity also occur.

Wrist, ankle and phalangeal joints are mainly affected, there is redness, pain and burning sensation in the affected joints. It is difficult to relieve from the disease .

Even though it gets relieved it repeatedly occurs in the same joints and produce ankylosis and hence all the movements of the joints become restricted. It may be associated with insomnia, restlessness and mild fever.

i) According to Yugi Muni,

"வைகிதமாய்க் கணைக்காலு முழங்கால் தானு
மற்கடஞ் சந்துபுற வடியும் வீங்கிச்
செய்கிதமாஞ் சிறுவிரல்கள் மிகவு நொந்து
சிந்தைதகு மாறியே சலிப்புண்டாகும்
பைகிதமாய் பயித்தியத்தில் வாத மிஞ்சிப்
பாரமா யுற்பவித் தழலுண் டாகும்
உய்கிதாம மசனமது தானும் வேண்டா
உதிரவாதச் சுரோணிதத்தி னுணர்ச்சி யாமே"

- சித்தமருத்துவம் (பொது) (பக்கம் எண்.609)

Vali azhal keel vayu, one of the 80 Vatha disease characterized by swellings especially in ankle, knee joints. Secondary it can produce depression, fatigueness, anorexia, because it is deranged (or) altered Vatha and Pitha dosham.

Vaidhya sara sankiragam ,

கை கால்கள் பொருத்துகளில் கரடுகட்டி மேனியெல்லாம்
தடித்துப் புண்ணாம் வாதபித்த சூலை எனப்படும்

Vatha pitha soolai cause ankylosis of the joints along with it some extra articular structure like skin may be involved with the formation of ulcers .

ii) According to Theriyar in Theriyar Vagadam,

"மொழி வாதம் மொழிகடோறுங் கரணைகட்டும்

.....மிசிந்து நோகும்"

- தேரையர் வாகடம் மூலமும், உரையும்

(பாடல் எண்.215, பக்கம் எண்.59)

Theriyar defines, Mozhi Vatham is one among the 81 Vatham. The characteristic features of joint pain, erosion and fusion of joints, causing deformity leading to inability to use the joints.

"முறிந்த வாதம் வந்தால்

எழுந்ததுமே நடக்க வொட்டாது"

- வாதநோய் மருத்துவம் (பக்கம் எண்.164)

"வாரிநீர் பெருகும் போலே உலர்ந்திடும் மூடடும், காலும்
பாகுற சந்து தோறும் பரந்துடல் சுளித்துக் குத்தும்

காரிகை உதிரமெல்லாம் நயந்துமே வருந்திவாடும்

கூரிய வாதமென்று கொற்றவர் வருத்தார் தாமே..."

- வாதநோய் மருத்துவம் (பக்கம் எண்.168)

Kooriya Vatham is one among the 80 types of Vatha disease. It is characterized by painful interphalangeal joints and swelling.

All the above features described in various texts closely resembles the clinical features of Vali Azhal Keel Vayu (Rheumatoid Arthiritis).

iii). According to Panditharathna Dr. S. Chidambarathanu Pillai, Siddha Medical Literature Research Centre,

"உடலது வெதும்பி கை கால்கள்

உளைவுடன் கடுத்து நொந்து

கடலலை தான் பிரண்டாப் போலே

கனத்துமே அயர்ந்து காணும்

சடமது விழுந்து தாகம்

சஞ்சலம் தோஷம் உண்டாய்

முடமதாம் கைகால் தன்னை

முடக்கிய வாதமென்றே''

DIAGNOSIS IN SIDDHA:

A). Piniyari Muraigal (Methods of Diagnosis) is based upon three main topics namely,

- Poriyal Aridhal (Physical Examination, Perception)
- Pulanal Aridhal (Palpation)
- Vinadhal (Interrogation)

Poriyal Aridhal (Inspection):

'Poriyal Aridhal' means examining the 'Pori' of the patient by the 'Pori' of the physician for proper diagnosis. Pori is considered as the 'five sensory organs' of perception namely.

- Mei (Skin)
- Vai (Tongue)
- Kan (Eye)
- Mooku (Nose)
- Sevi (Ear)

ஞானேந்திரிகளின் ஆய்வு

புலன்கள்	**தொழில்கள்**	**வளி அழல் கீல் வாயுவில் பாதிப்பு**
செவி	ஒலியை அறிய செய்ய	இயல்பு
மெய்	ஊடலில் ஊற்றை அறிதல்	மூட்டுக்களில் வலி, வீக்கம்
கண்	ஒளியை அறியச் செய்தல்	இயல்பு
நாக்கு	சுவையை அறியச் செயல்	இயல்பு
மூக்கு	வாசனையை நுகரச் செய்தல்	இயல்பு

கனமேந்திரியங்களின் ஆய்வு

புலன்கள்	தொழில்கள்	வளி அழல் கீல் வாயுவில் பாதிப்பு
வாய்	பேசச் செய்யும்	இயல்பு
கை	இடுதலும், ஏற்றலும் செய்யும்	பாதிப்பு
கால்	நடக்கச் செய்யும்	மூட்டுக்களில் வலி, நடக்கச் சிரமம்
எருவாய்	மலத்தைக் கழிக்கும்	மலச்சிக்கல்
கருவாய்	கரு, சுக்கிலத்தைக் கழிக்கும்	இயல்பு

Pulanal Aridhal (Palpation):

The five sense are given below:

- Smell
- Taste
- Vision
- Sensation of touch
- Hearing

By examining the 'Pulan' of the patient the physician can diagnose the disease.

Vinadhal (Interrogation):

Vinadhal is questioning and gathering information regarding the previous history of disease and clinical features which is much essential for diagnosis.

b). Envagai Thervugal (Eight Diagnostic Tools):

The excellent and unique method in the Siddha system is the Envagai Thervugal.

They are,

"தொடுக்கலுற்ற அட்டவிதப் பரிட்சை தன்னை

துலக்கமுறும் பண்டிதரே தெளி வதாகப்

பகுக்கரிய நாடியே நீபிடித்துப் பாரு

பகர்கின்ற வார்த்தைப் பார் நாவை பாரு

வகுக்கரிய தேகமெனத் தொட்டுப் பாரு

சகிக்கரிய மலத்தைப் பார் சலத்தை பாரு

சார்ந்த விழிதனைப் பார்த்து தெளிவாய் காணே''.

- அகத்தியர் வைத்திய வல்லாதி 600

1. Naadi (Pulse):

Among the Envagai Thervugal, Naadi is the most important. Naadi is felt as Vatham, Pitham and Kapham with the tip of the index, middle and ring fingers respectively over the end of the radius.

Normally Vatham, Pitham and Kapham are held in the ratio of 1:1/2:1/4. Derangement in this will reflect as disease. Naadi Nadai in Keel Vayu is,

வாதத்தின் குணமே தன்னில்

வயிறு பொருமிக் கொள்ளும்

தாகத்தில் மேனி கைகால்

சந்துமே கடுப்புத் தோன்றும்

- குறி யடையான் நாடி

அறியும் வாதத்தில் அடுத்த பித்தமாயின்

குறியதுதான் வாயுங்குழறும் நெறியாக

குளிருங் கால்வீங்கும் குடல் புரட்டும் விம்மித்

தெளிவில்லை புத்தியெனச் செப்பு

- ***கண்ணுசாமியம்என்னும்வைத்திய சேகரம்***

''வாட்டிடும் சேத்து மத்தில் வந்திடும் வாதமாகில்

நாட்டிய கால்கள் போல நரம்பெல்லாம் வலித்து நிற்கும்''

-(அ.மு.நாடி)

''திருத்தமாம் வாதத் தோடே தீங்கொடு பித்தஞ் சேரில்

பொருத்து கள்தோறும் நொந்து போதவே பிடிக்கும் சூலை''

நோயின் சாரம் - சித்தமருத்துவம் (பொது)

(பக்கம் எண்.634)

''வாய்த்திடும் வாதமிண்டு மொருசிலேத்ம மோடுமாகில்

உயத்திடும் கைால் நொந்து உயர்கனத் திடுப்புநோகும''

-(சதகநாடி)

''காணப்பா வாத மீறில்

கால்கைகள் பொருத்து நோகும்''

- காவியநாடி - சித்த மருத்துவம் (பொது)

(பக்கம் எண்.634)

In Vali Azhal Keel Vayu, the following Naadi is commonly felt.

- Vatham
- Vatha Pitham
- Pitha Vatham

2. Sparisam (Sensation to touch):

In Vali Azhal Keel Vayu, heat is noticed over the affected joints.

3. Naa (Tongue):

In Vali Azhal Keel Vayu, no abnormality is seen in Naa.

4. Niram (Colour):

In Vali Azhal Keel Vayu, some skin colour changes are seen in affected area due to inflammatory mechanism.

5. Mozhi (Voice):

In Vali Azhal Keel Vayu, no abnormality is seen in mozhi.

6. Vizhi (Eyes):

In Vali Azhal Keel Vayu, Eye pallor is reported in some cases.

7. Malam (Faeces):

In Vali Azhal Keel Vayu, Constipation is reported in some cases.

8. Moothiram (Physical appearance of Urine):

In urine, Neer kuri and Nei kuri examinations are done.

Nei kuri (Oil Examination):

Prior to the day of urine examination the patient is instructed to take a balanced diet and quantities of food must be proportionate to his / her appetite. The patient should have no disturbed sleep. After waking up in the morning, the first urine voided is collected in a clear wide mouthed glass dish or china clay bowl and is subjected to analysis of Neer kuri and Nei kuri within one and a half an hour of its collection.

The collected urine specimen is kept in a glass dish or china clay container and observed under direct sunlight without shaking the vessel. Then add one drop of gingelly oil and observe the spreading pattern and conclude as follows:

"அரவென நீண்டின அ.ஃதே வாதம்

ஆழிபோற் பரவின் அ.ஃதே பித்தம்

முத்தொத்து நிற்கின் மொழிவ தென் கபமே"

- நோய் நாடல் பாகம்.1

(பக்கம் எண்.298, 299)

Neer kuri:

" வந்த நீர்க்கரி யெடை மணம் நுரை எஞ்சலென்

றைந்தியலுளவை யறைகுது முறையே"

- சித்த மருத்துவாங்க சுருக்கம்

(பக்கம் எண்.510)

In urine examination the following characteristic features were observed namely.

Niram - Colour

Edai - Specific gravity

Manam - Smell

Nurai - Frothy nature

Enjal - Quality of urine voided

Apart from these, frequency of micturition, abnormal constituents such as sugar, protein, blood stains, pus, crystals also to be found out.

In Vali Azhal Keel Vayu, Straw or Hay coloured urine was noticed in Neer kuri.

c. பருவகாலம்:

வ.எண்	குற்றம்	காலம்
1.	வாதம் தன்னிலை அடைதல்	முன்பனி காலம் ,பின்பனி காலம், கூதிர் காலம், இளவேனில் காலம்
2.	வாதம் தன்னிலை வளர்ச்சி	முதுவேனில் காலம்
3.	வாதம் வேற்றுநிலை வளர்ச்சி	கார்காலம்

முதுவேனிற் காலத்தில் நமது உடலில் வறட்சி ஏற்பட்டு வளிநோய் வருவதற்கு ஏதுவாகிறது.

d). திணை (Geographical distribution):

குறிஞ்சி : மலையும், மலை சார்ந்த பகுதியும்

முல்லை : காடும், காடு சார்ந்த பகுதியும்

"முல்லை நிலத்தயமே மூரிநிரை மேனினுமவ்

வெல்லை நிலைத்தபித்த மெங்குறுங்காண் - வல்லையெனின்

வாதமொழி யாததனுண் மன்னு மவைவழிநோய்ப்

பேதமொழி யாதறையப் பின்பு"

(பொழிப்புரை):

முல்லை நிலத்தில் ஆடு, பசு, எருமை முதலியவை இருப்பினும் அந்நிலத்திற்கு உரிய பித்தநோய் எவ்விடத்திற் போய் நிற்கும்? அன்றியும் வல்லைநோய் உண்டாகும். இதனால் வாத நோயும் வந்துறும் ஆகையால் இந்நிலத்தில் வாதச் சார்பான நோய்கள் பலவும் உண்டாகும்.

மருதம் : வயலும், வயல் சார்ந்த பகுதியும்

நெய்தல் : கடலும், கடல் சார்ந்த பகுதியும்

"நெய்தனிலே மேலுப்பை நீங்கா துறினுமது

நெய்தனிலே மேதங்கு வீடாகும் - நெய்தல்

மருங்குடலை மிக்காக்கும் வல்லுறுப்பை வீக்கும்

கருங்குடலைக் கீழிறங்குங் காண்."

(நெய்தல் நிலத்தில்இமிக்க உப்பு விளை பொருளாயிருப் பினும் அந்நிலத்தில் கொடுமையான வாதநோய்களே உண்டாகும்)

பாலை : மணலும், மணல் சார்ந்த பகுதியும்

முல்லை மற்றும் நெய்தல் நிலங்களில் வாத நோய்கள் பெருமளவில் ஏற்படும்.

e). ஏழு உடல் தாதுக்களின் ஆய்வு (Seven Udal Thathukal Examinations):

Seven Thathus

1. Saaram – Strengthens the body and mind
2. Senner – gives power, knowledge and boldness to the mankind
3. Oon – It strengthens the body
4. Kozhuppu – It lubricates the joints
5. Enbu – It protects all the internal organs and gives the structure to the body
6. Moolai – It is present in the bones
7. Sukkilam and suronitham- Mean for reproduction (male and female gonads)

SI.No.	Udal Thathukal	Increased conditions	Decreased condition
1.	Saaram	Loss of appetite , excessive salivation	Tiredness , fatigue , diminished activity of the sense organs.
2.	Senneer	Boils and tumours in different parts of the body, spleenomegaly, colic pain, increased blood pressure reddish eyes and skin, jaundice, leprosy, haematuria.	Tiredness, lassitude, anemia.
3.	Oon	Tumours or extra growth around the neck, face, abdomen, thigh, genitalia etc., with dyspnoea. Muscle wasting	Muscle wasting
4.	Kozhuppu		Pain
5.	Enbu	Extra growth of bone and teeth	Weak bones , teeth , nails and hair.
6.	Moolai	Heaviness, swollen eyes, swollen phalanges, oliguria and non healing ulcers. Osteoporotic changes.	Osteoporotic changes.
7.	Sukkilam or Suronitham	Increased sexual activity and symptoms as that of urinary calculi.	Infertility , pain in genitalia.

In Vali Azhal Keel Vayu:

Saaram, Seener, Kozhuppu, Enbu, Moolai thathukal are commonly affected.

Saaram : Weakness, pain in all major and minor joints.

Seneer: Tiredness anemia

Kozhuppu : Early morning stiffness occurs in affected joints.

Enbu : Joint space narrowing, marginal erosions and deformities.

Moolai : Joint effusion and oedema are seen in the joints.

MUKKUTRAM:

Uyir thathukal i.e., Vatham, Pitham and Kapham responsible for normal physiological conditions of the body. Vatham is mainly responsible for proper locomotor functions. Bones and joints are the major site of Vatha.

Vatham:-

Locations of Vatham

Vatham is located in abana vayu , faeces , idakalai , spermatic cord , pelvic bone , skin, nerves, joints etc.,

Types of vatham

1. Piranan (uyirkkal)

This controls knowledge, mind and five objects of senses which are helpful for breathing and digestion

2. Abanan (Keezh Nokkukal)

This is responsible for all downward movements such as passing of urine, stools, sperm, menstrual flow, etc.,

3. Samanan (Nadukkal)

This aids for proper digestion.

4. Viyanan (Paravukkal)

This is responsible for movements of all parts of the body

5. Uthanan (Mel Nokukkal)

Responsible for all upward visceral movenments such as cough, hiccup, vomiting, nausea etc.,

6. Nagan

Responsible for opening and closing of the eyes.

7. Koorman

Responsible for vision and yawning

8.Kirukaran

Responsible for salivation, nasal secretion and appetite

9. Dhevathathan

Responsible for laziness, sleep and anger

10.Dhananjeyan

Produces bloating of body after death and escapes on the third day by bursting the cranium

In vali azhal keel vayu Abanan, Viyanan, Samanan, Koornan and Kirukaran are affected and they produce symptoms as follows

- Affected Abanan produces constipation,
- Affected Samanan produces loss of appetite and indigestion
- Affected Viyanan produces pain and restriction of movements of the Affected joints
- Affected Koorman produces disturbed sleep
- Affected Kirukaran produces loss of appetite

Pitham

Locations of Pitham

Pitham is located in pirana vayu, bladder, moolaakini, heart, umbilical, region, abdomen, stomach, sweat glands,eyes, saliva, blood etc.,

Types of pitham

Analapitham- It gives appetite and helps digestion

Pirasagapitham – It gives complexion to the skin

Ranjagapitham – It gives colour to the blood

Alosagapitham – It brightens the eye

Sathagapitham – it controls the whole body

In vali azhal keel vayu Analapitham, Pirasagapitham, Ranjagapitham, and Sathagapitham maybe affected.

Affected Analapitham produces loss of appetite

Affected Ranjagapitham produces anemia

Affected Sathagapitham produces disability to carry out regular works

Affected Pirasagapitham produces pallor of skin

Kabam

Kabam is located in samana vayu , sperm, head, tongue, vulva etc.,

Types of Kabam

Kilethagam – Lies in stomach, makes food soft and helps in digestion

Avalambagam – Lies in lungs, controls the heart and other kabas,

Pothagam – responsible for indentifying tastes

Tharpagam – present in the head and responsible for the coolness of both eyes

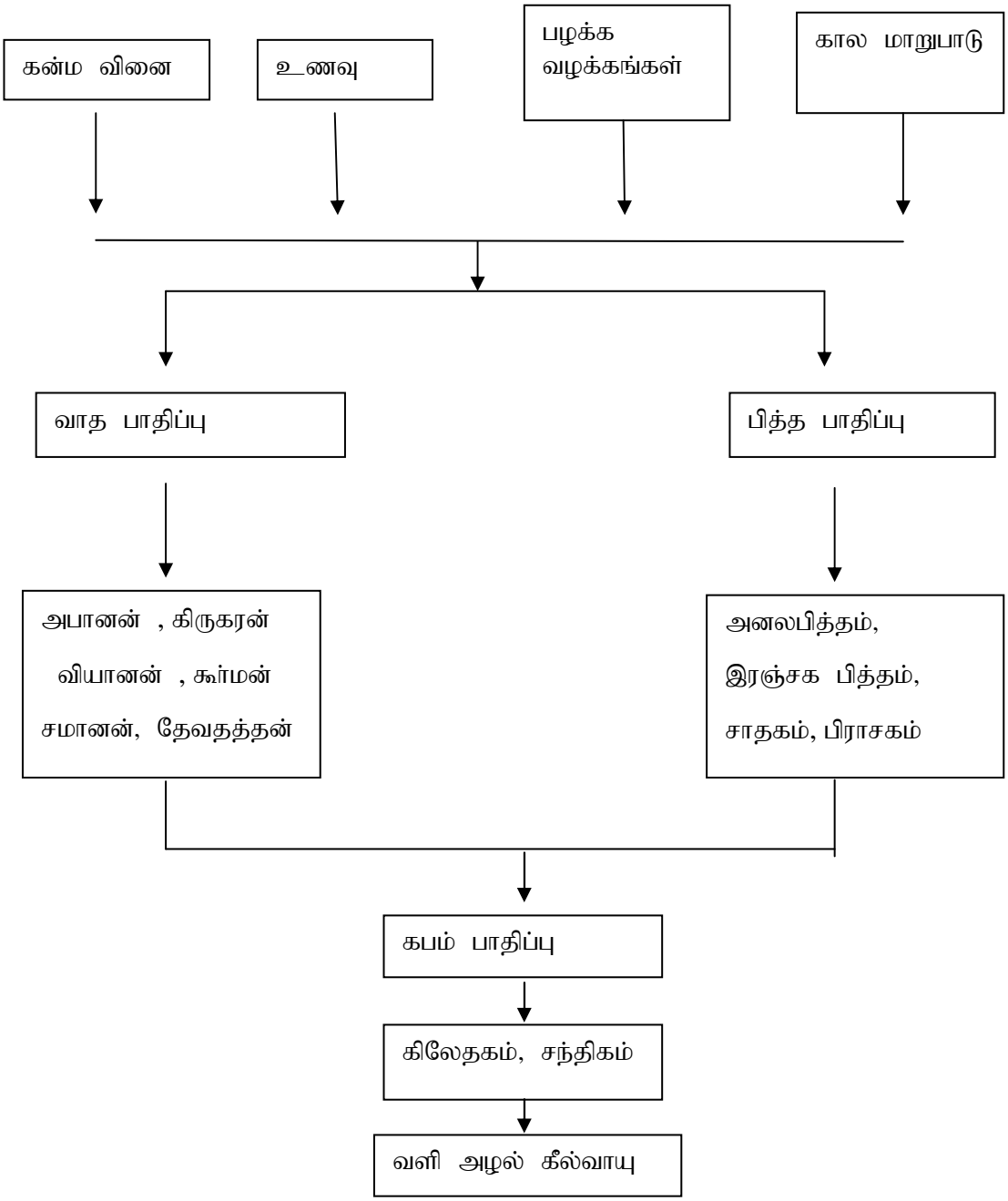
Santhigam – responsible for the lubrication and free movement of the joints.

In vali azhal keel vayu, Kilethagam and Santhigam may be affected

Affected Kilethagam causes loss of appetite

Affected Santhigam causes pain in the joints .

Noi Varum Vali (Aetiology) is tabulated below:



In Vali Azhal Keel Vayu, the Vatha kutram and Pitha kutram is mainly affected. The symptoms and signs are,

- Deranged Viyanan leads to painful stimuli and difficulty in movements with early morning stiffness.
- Deranged Abanan leads to constipation.

- ❖ Inflammatory changes of the joints, heat, redness and swelling are developed due to altered Pitham. Ranjaga Pitham is reduced haemoglobin level. Sathaga Pitham is hindering of the locomotor functions.
- ❖ Along with Vatham, Kapham is also deranged, Santhigam is affected and this leads to abnormality in joint movements.
- ❖ Erosion, loss of joint space, increased secretion of synovial fluid may lead to synovial effusion due to increased Kapham.

SI.No	Name	Location	Physiological function
1.	Abanan	Lower abdomen and extremitis	Responsible for urination, defecation, parturition, menstruation and ejaculation of the sperm.
2.	Viyanan	Heart, Skin	Responsible for movements of all parts of the body and sensation.
3.	Samanan	Stomach	Responsible for proper digestion.

NOI KANIPPU VIVATHAM (Differential Diagnosis):

Vali Azhal Keel Vayu is differentiated from the following diseases,

1. சந்து வாதம்(Santhu Vatham) (Infective Arthritis)

"செய்கை தான் சந்துகளு மிகத் திமிர்ந்து
சடமெங்கும் நொத்துமே மிகவ ழற்றி
நைகையாய் நலுத்துமே மயிர்க் ச்சலிட்டு
நாணியே முன்போல் நடை கொடாது
மைகைதான் மயக்கமொடு வாய் நிருறும்
வரண்டிடுமே நாவுதானடிக்க டிக்குக்
கைகால்தான் தரணிதனிற் றரிக் கொணாது
சஞ்சரிக்குஞ் சந்துவாம் வாதங் கேளே".

Pain in joints ,body pain, inability to walk, giddiness,dryness of tongue, excessive salivation and unable to stand in floor is a characteristic features of the disease.

2. Azhal Keel Vayu: (Osteoarthritis)

"பித்தக்கல் வாயவு தன்னாற் பிறங்குகீன் முட்டு வீங்கிச்

சித்தர் மருத்து வத்துஞ் சீரபடாத் தன்மைத் தாகித்

தத்தறு காய்ச்சல் கண்டு சாலவே தனைதான் தந்தே

மெத்தறு சிகிச்சை தன்னால் மென்மெல நீங்கு மப்பா"

- சபாபதி கையேடு சித்த மருத்துவம் (பொது)

(பக்கம் எண்.626)

It is characterized by swelling of joint associated with severe pain and fever. In later stages joints gets fused, resulted in disability.

3. Iya Keel Vayu: (Tubercular arthritis)

"கருதருங் கபக்கில் வாயு கண்டிடின் உடலிளைக்கும்

உருமெலிவாக்குங் கொள்ளும் உண்டியைச் சுருக்கு மினபந்

தருதுயில் நங்கு முட்டிற் றாங்கொணா வலுவையாக்கும்

இருமலே விக்கல் வாந்தி, சோபை பாண்டெழுப்பும் பாரே"

- சபாபதி கையேடு சித்த மருத்துவம் (பொது)

(பக்கம் எண்.627)

It is characterized by severe pain in the joints associated with loss of weight, anorexia, insomnia, cough, hiccough, vomiting, anemia and dropsy.

4. நரித்தலை வாதம் (Narithalai Vatham) (Gonococcal arthritis)

"முர்க்கமாய் முயன்று முழங்கால்தான் விங்கி

முதிர்ந்துரத்த முந்திரண்டு முயற்சி யாகி

நிக்கமாய் தின்றிடவொ ணாமற் றானும்

நிமிர்ந்திடுகில் சந்துந்தான் முடக் கொணாமல்

செழுமை நரித் தலை போல மிகவே விங்கி

நார்க்கமாய் நாடியுமே படபடக்கும்

நரித்திலையின் வாதமென்றே நவில லாமே''.

Swelling of the knee joint with hyperemia, difficulty to stand,palpitation will be seen, the swelling looks like the head of the fox are the features of this disease.

5. Vali Iya Keel Vayu: (Synovial effusion)

''அவையம் வாதக் கபக்கீல் வாயுவான் வலி மிகுந்தே

உயங்கு நீர் கோத்து கீல்கள் ஒரியின் தலைபோற் காணும்

நயங்கொள்ள முடக்கல் நீட்டல் நண்ணிடாமெய் யுங்காயும்

மயக்குறு முறக்கமின்னாம் மன்னிய நெரிக்கட் டாமே''

- சபாபதி கையேடு சித்த மருத்துவம் (பொது)

(பக்கம் எண்.628)

It is characterized by pain in the joints associated with effusion of joint fluid, swelling, restricted joint movements, pyrexia, fainting, insomnia, asymmetrical presentation lymphadenopathy, generalized malaise, atrophy of the affected limb etc.,

MODERN ASPECTS

RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) is a chronic inflammatory, autoimmune disease where the body's immune system attacks normal joint tissues, causing inflammation of the joint lining. This inflammation of the joint lining can cause pain, stiffness, swelling, warmth, and redness that can eventually result in bone erosion and joint deformity.

EPIDEMOLOGY:

Rheumatoid arthritis is affecting approximately 1.3 million people in the developed countries. The disease is three times more common in women as in men. The individuals with HLA-D4 and HLA-DR4 are more prone to Rheumatoid arthiritis. It afflicts people of all races equally. The disease can begin at any age and even affects children (juvenile idiopathic arthritis), but it most often starts after 40 years of age and before 60 years of age. Though uncommon, in some families, multiple members can be affected, suggesting a genetic basis for the disorders.

CAUSES AND RISK FACTORS:

The cause of rheumatoid arthritis is unknown.

The risk factors may be:

- Infections.
- Hereditary.
- Environmental factors.

  - Smoking
  - Tobacco chewing
  - Exposure to Silica Mineral
  - Periodontal diseases

Even though infectious agents such as **viruses, bacteria,** and **fungi** have long been suspected, none has been proven as the cause. The cause of rheumatoid arthritis is a very active area of worldwide research.

Rheumatoid arthritis is a chronic multi system, inflammatory, autoimmume disease that affects the synovia and cartilages of small and large joints as well as other organ system.It is a destructive disease that involves boyh cell mediated and humoral immune responses.

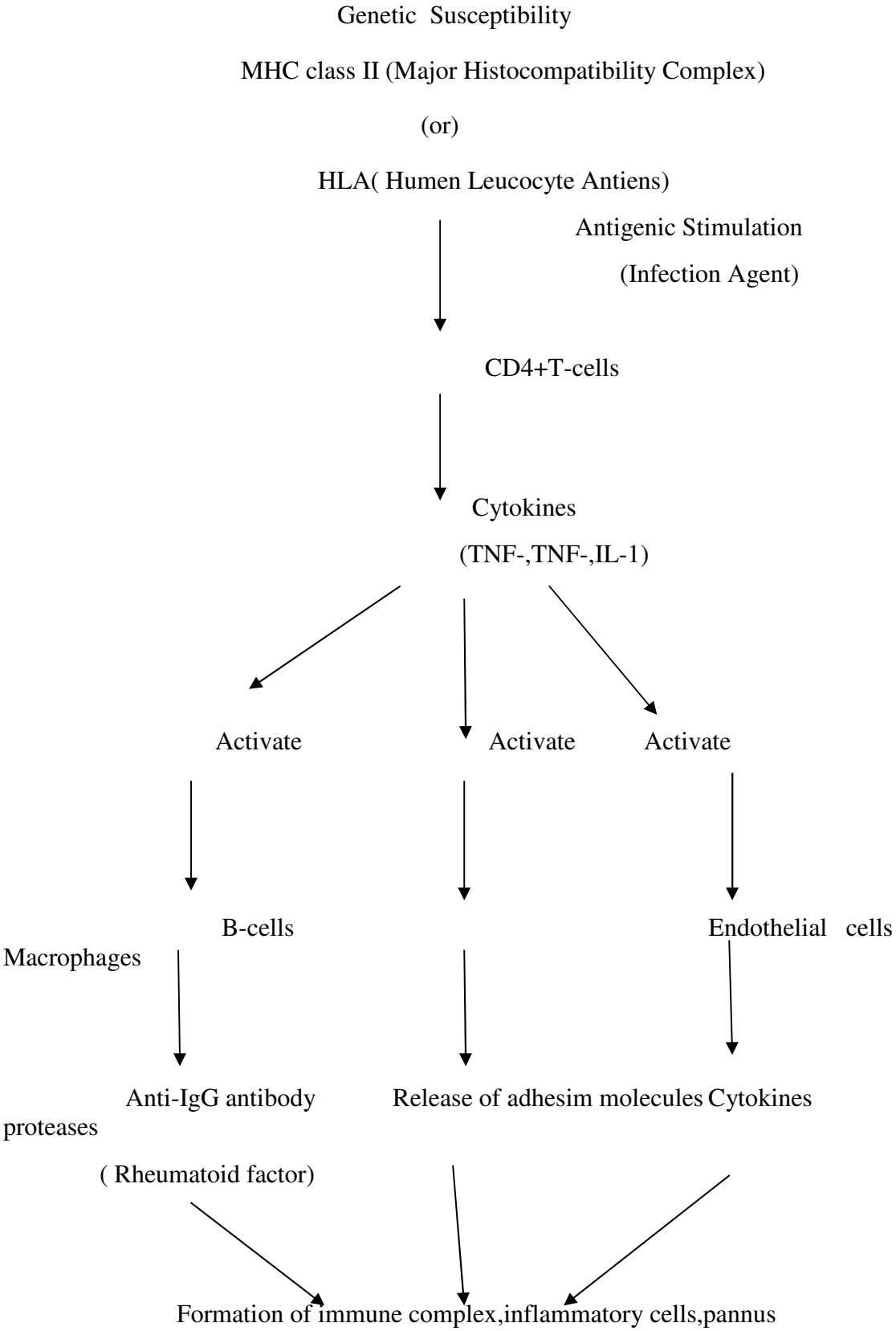
The initial underlying causes of RA is still unknown. Cartilage damage is because of the CD4+ T-cells recognition of antigens with in the joint that triggers the release of inflammatory cytokines that lead to the accumulation of neutrophils and macrophages.

Within the inflamed synovia or B cells,plasma cells,CD4+ T cells, and various types of inflammatory cytokines,such as tumor necrosis factor(TNF),IL-1,IL-8,and IFN-α

Rheumatoid factors are formed that facilitate the formation of immune complexes.In the most patients with RA, antibodies to cyclic citrullinated peptide (anti-CCP) are detected before RA symptoms develop.

In advanced stages of RA,deposition and complement fixation of the immune complexes may contribute not only to joint destruction but also to vasculitis,carditis,and pleuritis.

PATHOGENESIS:



Synovium:

The synovium, in normal joints, is a thin delicate lining that serves several important functions. The synovium serves as an important source of nutrients for cartilage since cartilage itself is a vascular. In addition, synovial cells synthesize joint lubricants such as hyaluronic acid, as well as collagens and fibronectin that constitute the structural framework of the synovial interstitium.

1. **Synovial lining or intimal layer:** Normally, this layer is only 1-3 cells thick. In RA, this lining is greatly hypertrophied (8-10 cells thick). Primary cell populations in this layer are fibroblasts and macrophages.

2. **Subintimal area of synovium:** This is where the synovial blood vessels are located this area normally has very few cells. In RA, however, the subintimal area is heavily infiltrated with inflammatory cells, including T and B lymphocytes, macrophages, macells,andmononuclear cells that differentiate into multinucleated osteoclasts.

The intense cellular infiltrate is accompanied by new blood vessel growth (angiogenesis). In RA, the hypertrophied synovium (also called pannus) invades and erodes contiguous cartilage and bone. As such, it can be thought of as a tumor-like tissue, although mitotic figures are rare and of course, metastasis does not occur.

Cartilage:

Composed primarily of type II collagen and proteoglycans, this is normally a very resilient tissue that absorbs considerable impact and stress. In RA, its integrity, resilience and water content are all impaired. This appears to be due to elaboration of proteolytic enzymes (collagenase, stromelysin) both by synovial lining cells and by chondrocytes themselves.

Cytokines including IL1 and TNF drive the generation of reactive oxygen and nitrogen species and while increasing chondrocyte catabolic pathways and matrix destruction, also inhibit new cartilage formation. Polymorphonuclear leukocytes in the synovial fluid may also contribute to this degradative process.

Bone:

Composed primarily of type I collagen, bony destruction is a characteristic of RA. This process is primarily driven by the activation of osteoclasts. Osteoclasts differentiate under the influence of cytokines especially the interaction of RANK with its ligand. The expression of these are driven by cytokines including TNF and IL1, as well as other cytokines including IL-17. There may also be a contribution to bony destruction from mediators derived from activated synovial cells.

Synovial Cavity:

The synovial cavity is normally only a "potential" space with 1-2ml of highly viscous (due to hyaluronic acid) fluid with few cells. In RA, large collections of fluid ("effusions") occur which are, in effect, filtrates of plasma (and therefore, exudative – i.e., high protein content). The synovial fluid is highly inflammatory. However, unlike the rheumatoid synovial tissue in which the infiltrating cells are lymphocytes and macrophages but not neutrophils, in synovial fluid the predominant cell is the neutrophil.

Disease Initiation:

a) Genetic Susceptibilities:

Class II MHC on the surface of an antigen presenting cell interacts with a T cell receptor in the context of a specific antigen, usually a small peptide sequence from a protein. A sequence of amino acid residues with highly conserved sequence and charge characteristics within the hypervariable region of HLA-DR4 remains the largest genetic risk factor described for RA, estimated to contribute approximately 30% of the genetic risk for the disease. It is hypothesized that a triggering peptide (or peptides) with a tight conformational fit for the pocket formed by these residues is an early event leading to the activation of T lymphocytes. More recently, it has been found that modified citrullinated peptides may have significant binding specificity for shared epitope alleles, with some data now suggesting that citrullinated sequences from different proteins are associated with allelic restriction.

Other genetic susceptibilities include peptidyl arginine deiminase-4 (PAD-4) which may lead to increased citrullination, PTNP22, STAT4, and CTLA4 which may be involved in T cell activation, TNF receptors, and others. That RA has a genetic component is also borne out through monozygotic (from the same embryo, thus nearly identical DNA) and dizygotic (from different embryos) twins. In Research studies the concordance rates between twins was higher in monozygotic twins ranging from 15-35% compared with dizygotic twins in which the concordance was in the 5% range. Even the dizygotic RA prevalence was higher than the general population estimates of approximately 1%. It is important to emphasize however that even in twins with nearly identical DNA, there was far from perfect correlation of the development of RA, implicating many other factors related to the development of disease than genetic factors.

b) Citrullination:

Citrulline is a post-translational modification that occurs on arginine residues contained within proteins and peptides. There are a number of enzymes that can cause citrullination to occur, present in various cell types and tissues known as Peptidyl Arginine Deiminases (PADs). Citrullination is a normal process, required for normal skin formation and other physiologic functions. The triggering factors of RA results in increase CITRULLINATION of Proteins. However, in rheumatoid arthritis an autoimmune response develops against citrullinated peptides detected as anticitrullinated peptide antibodies (ACPA).

The mechanisms to citrullination that lead to RA remain unclear. A polymorphism in the PAD4 gene which may lead to increased citrullination has been described in few populations. In RA patients, autoantibody responses also develop against the PAD4 protein, associated with a more aggressive disease course. One species of oral bacteria Porphyromonas gingivalis has a PAD enzyme. Given the relationships described with periodontal disease and RA, it has been hypothesized that this bacteria may also serve to initiate citrullination in the preclinical phases of RA.

c) Propagation of Disease:

i) T cell activation

Upon encounter with antigen in the context of MHC on an antigen presenting cell, a T lymphocyte is positioned for 3 possible fates: activation, anergy /tolerance, or apoptosis (death). T cell activation is only possible if the T cell receives a "second signal" through engaging additional cellular receptors. One of the most important of these second signals is delivered through the CD28 molecule on the surface of the T cell but many other second signals are involved in this process of "costimulation". Upon engagement of these receptors, a T cell usually becomes activated. Failure to engage the stimulatory receptors, or engagement of a downregulator receptor will cause the cell to become tolerant to the antigen or to undergo programmed cell death through apoptosis. The process of T-cell costimulation is interrupted by abatacept, a biological therapy used to treat RA.

When T cells become activated, they will in turn proliferate and begin to secrete additional cytokines including IL-2 which furthers their proliferation, and depending on other exposures, cytokines such as IFN-γ, TNF, and IL-4. It is the effect of these T-cell derived cytokines that additional cells become activated. T cells also directly interact through surface receptors with other cells to generate additional activation signals.

ii) B Cell Activation and Autoantibodies

B cells become activated through interactions with T cells and through soluble cytokines that enhance their proliferation and differentiation. B cells express a number of receptors on their surfaces during their differentiation, including the molecule CD20, which is lost upon terminal differentiation to antibody-forming plasma cells. B cells and plasma cells can be found in rheumatoid synovium sometimes as lymphoid aggregates in the subsynovium. The effects of B cells extend beyond their roles in forming plasma cells including cytokine production, direct cellular interactions, and they themselves serve as antigen-presenting cells to T lymphocytes. The role of B cells in RA has been clearly demonstrated with the efficacy of rituximab which eliminates circulating B cells, though with limited impact on autoantibody formation.

Rheumatoid arthritis is characterized by the presence of autoantibodies known as rheumatoid factors (RF) and anti-citrullinated peptide antibodies (ACPA, which includes the anti-cyclic citrullinated peptide antibody or anti-CCP).These are autoantibodies in the classical sense; they are antibodies directed against native antibodies, most classically described as IgM antibodies that recognize the Fc portion of IgG molecules, but RF may also be of the IgG or IgA isotypes.

iii) Effector Cell Activation

While T cells and B cells represent the immunological aspects of RA, most of the damage from the disease is driven through effector cells and their products including cytokines and other mediators. The synovial lining in RA represents an expansion of fibroblast like cells and macrophages. It is the macrophage that has been seen as one of the master orchestrators of the effector damage in RA. Macrophages are rich sources and major producers of pro inflammatory cytokines including TNF, IL-1, IL-6, IL-8, and GMCSF. These cytokines further stimulate the macrophage, as well as other cells in the microenvironment in a including fibroblasts and osteoclasts, and finally at distant sites in the body through cell surface receptors including the hepatocyte which is responsible for the generation of acute phase response proteins (such as C-reactive protein). Macrophages are also producers of prostaglandins and leukotrienes, nitric oxide, and other pro-inflammatory mediators with local and systemic effects. The synovial fibroblast also secretes cytokines including IL-6, IL-8 and GM-CSF, and other mediators including destructive proteases and collagenases.

- **Neutrophils** are recruited in very large numbers to the rheumatoid cavity where they can be aspirated in the synovial fluid. The recruitment of neutrophils to the joint is likely driven by IL-8, leukotriene B4, and possibly localized complement activation through C5a. Neutrophils in the synovial fluid are in an activated state, releasing oxygen-derived free radicals that depolymerize hyaluronic acid and inactivate endogenous inhibitors of proteases, thus promoting damage to the joint.

- **Chondrocytes** like synovial fibroblasts, are activated by IL1 and TNF to secrete proteolytic enzymes. They may, therefore, contribute to the dissolution

of their own cartilage matrix, thus explaining the progressive narrowing of joint spaces seen radiographically in this disease.

d) Inflammatory Mediators in RA:

Cytokines:

One of the most important group of mediators in RA are cytokines. The most prominent of these are TNF, IL-1, and IL-6. These cytokines, released in the synovial micro environment have autocrine (activating the same cell), paracrine (activating nearby cells), and endocrine (acting at distant sites) effects and accounting for many systemic manifestations of disease. There are many shared functions of TNF, IL-1, and IL-6, and these cytokines in turn upregulate the expression of the others. Among the important effects of these cytokines are:

- Induction of cytokine synthesis
- Upregulation of adhesion molecules
- Activation of osteoclasts
- Induction of other inflammatory mediators including prostaglandins, nitric oxide, matrix metalloproteinases
- Induction of the acute phase response (Increased C-reactive protein, increased ESR)
- Systemic features (Fatigue, fever, Cachexia) Activation of B cells (IL-6)

Soluble mediators of inflammation that may diffuse in from blood and/or be formed locally within the joint cavity include **prostaglandins, leukotrienes, matrix metalloproteinases.** Prostaglandins are involved in pain sensitization, localized inflammation and some effects on bone. Leukotrienes play roles in vascular permeability and chemotaxis. Matrix metalloproteinases (MMPs) are potent in their ability to enzymatically degrade the collagen matrix of cartilage. Kinins cause release of prostaglandins from synovial fibroblasts, and are also potent algesic (painproducing) agents.

Complement may be available for interaction with immune complexes to generate additional chemotactic stimuli. The neuropeptide substance P is a potent vasoactive, pro inflammatory peptide that has also been implicated in RA.

SIGNS AND SYMPTOMS:

- RA symptoms come and go, depending on the degree of tissue inflammation.
- When body tissues are inflammed, the disease is active.
- When tissue inflammation subsides, the disease is inactive (in remission).
- Remissions can occur spontaneously or with treatment and can last weeks, months, or years.
- During remissions, symptoms of the disease disappear, and people generally feel well.
- When the disease becomes active again (relapse), symptoms return.
- The return of disease activity and symptoms is called a **Flare.**
- The course of rheumatoid arthritis varies among affected individuals, and periods of flares and remissions are typical.

When the disease is active, RA symptoms can include

- Fatigue
- Insomnia
- Lack of appetite.
- Low-grade fever
- Muscle and joint pain
- Stiffness

Muscle and joint stiffness are usually most notable in the morning and after periods of inactivity. This is referred to as morning stiffness and post-sedentary stiffness. Arthritis is common during disease flares. Also during flares, joints frequently become warm, red, swollen, painful, and tender. This occurs because the lining tissue of the joint (synovium) becomes inflammed, resulting in the production of excessive joint fluid (synovial fluid). The synovium also thickens with inflammation (synovitis).

Rheumatoid arthritis usually inflammes multiple joints and affects both sides of the body. In its most common form, therefore, it is referred to as a **symmetric polyarthritis.** Early RA symptoms may be subtle. The small joints of both the hands and wrists are often affected. Symptoms in the hands with rheumatoid arthritis include difficulty with simple tasks of daily living, such as turning door knobs and opening

jars. The small joints of the feet are also commonly involved, which can lead to painful walking, especially in the morning after arising from bed.

Chronic inflammation can cause damage to body tissues, including cartilage and bone. This leads to a loss of cartilage and erosion and weakness of the bones as well as the muscles, resulting in joint deformity, destruction, and loss of function. Rarely, rheumatoid arthritis can even affect the joint that is responsible for the tightening of our vocal cords to change the **tone of our voice**, the cricoarytenoid joint. When this joint is inflammed, it can cause **hoarseness of the voice**. Symptoms in children with rheumatoid arthritis include limping**, irritability, crying, and poor appetite.**

Extra- articular manifestations of Rheumatoid Arithiritis

Systemic

- Fever
- Fatigue
- Weight loss
- Susceplibility to infection

Musculoskeletal

- Muscle wasting
- Bursitis
- Tenosynovitis
- Osteoporosis

Haematological

- Anamia - NSAID – GIT blood loss leads to anaemia
- Eosinophila
- Thrombocytosis

Lymphatic

- Felty's syndrome
- Spleenoonegely

Nodules

- Scleritis

Vasculitis

- Digital arteritis
- Venous ulcer
- Visceral arteritis
- Pyoderma gangrenosum
- Mono Neuritis Multiplex

Cardiovascular system

- Pericarditis
- Myocarditis
- Endocarditis
- Conduction defects
- Coronary vasculitis
- Granulomatous aortitis

Pulmonary system

- Nodules
- Plereral offusions
- Caplan's syndrome
- Bronchiolitis
- Fibrosing alveotitis

Central Nervous system

- Cervical and compression
- Compressive myelora radiculopathy
- Periphral neuropathies
- Mono Neuritis multiplex

Amyloidosis is a rare complication of prolonged active disease and usually presents with Nephrotic syndrome.

Subcutaneous Rheumatoid Nodules

Subcutanneous and intracutaneous nodules are the hall mark of the disease.

It develop in 20 to 30% of patients with Rheumatoid Arthritis.

They are usually found on peri articular structures,extensor or other areas subject to Mechanical pressure.

Common locations include a olecranon bursa, the proximal ulnar, the achilles tendon, the occipital bone etc., They are also found in the flexor tendon,the scalar,with in the aortic value,myocardium,larynx and vocal cord.

Histologically, the nodules consist of a central zone of necrotic material,including collagen fibrils,non-collagenous filaments and cellular debris,mid zone of palisading macrophages that express HLA-DR antigens and an outer zone of granuloma tissue.

Rheumatoid vasculitis

It can affect nearly any organ system and is seen in patients with severe from and high titres of circulating rheumatoid factors.

Neurovascular disease presenting either as distal sensory neuropathy or as mononeuritis multiplex may be the only sign of vasculitis.

Custaneous vasculitis usually presents as crops of small brown spots in the nail bed, nail folds and digital pulp,large ischaemic ulcers especially in the lower extremity may also develop.

Vasculitis also involves the lungs,bowel,liver,spleen,pancreas,lymphnodes and testes.

Renal Involvement

Renal papillary necrosis and interstitial nephritis occasionally occur IgA nephropathy associated with elevated serum levels of IgG and IgA is described in Rheumatoid Arthritis.

Liver Involvement

This is evident in about 10% of patients with active disease.

There may be mild hepatospleenomegaly and asympatamatic elevation of the serum alkaline phosphatase.

Kupffer cell hyperplasia and lymphocytic infilteration of the portal tracts may be seen.

Pulmonary Manifestations

This is more in men.

Pleuro pulmonary nodule may occur as singly or in clusters when they appear in individual with the pneumoconiosis and diffuse nodular fibrotic nodules 0.5-5 cm in diameter are seen mainly in the periphery of the lung fields. This association is known as **Caplan's syndrome.** These nodules may produce pneumothorax or broncho pleural fistula.

Interstitial fibrosis.

- Pleurisy and pleural effusion produce frank synovitis.
- Pulmonary fibrosis is common in rheumatoid arthritis but is often subclinical.
- Pulmonary hypertension due to vasculitis.
- Obliterative bronchiolitis is a rare but rapidly progressive and fatal process.
- Cardiovascular Manifestations
- Asymptamatic pericarditis
- Pericardial effusion
- Constructive pericarditis
- Cardiomyopathy
- Coronary artery occlusion
- Acute aortic regurgitation
- Valvulitis

Hematological Manifestations

Felty's syndrome

This syndrome describes the association between rheumatoid arthritis, spleenomegaly and leucopenia with normochromic normocytic aneamia, thrombocytopenia,lymphadenopathy,cutaneous pigmentation, chronic skin ulceration and weight loss.

Thrombocytosis

Eosinophilia

Neuro Muscular Mainfestations

Peripheral neuropathies - usually sensory or occasionally motor.

Atlanto axial or mid cervical spine subluxation may produce Vertibro Basilar Insufficiency(VBI) and neurological manifestation due to direct compression of the cord.

Entrapement neuropathy e.g. Median never compression at wrist – **carpal tunnel syndrome.**

Posterior tibial nerve compression at ankle – **Tarsal tunnel syndrome.**

Ulnar nerve compression at elbow.

Cervical myelo radiculopathy.

Tenosynovitis and Bursitis

"Triggering" of the fingers may be associated with nodules in the flexor tendon sheaths which can progress to permanent flexion contractures or tendon rupture if left untreated.

Muscular Changes

Muscle atrophy in Rheumatoid patients is usually attributed to reflex inhibition and disuse because of articular inflammation.

Ocular manifestations

Episcleritis which is mild and transient.

Scleritis which involves the deeper coat of the eye and is a more serious inflammatory condition.

Keratolysis(corneal melting)

Sclero malacia

Sclero malacia perforans.

Sjogren's syndrome – Keratoconjunctivitis sicca, Xerophthalmia and Rheumatoid Arthritis or other connective tissue disorder with the lack of tear and salivary secretions.

The symptoms are gritty sensations in the eyes, dryness of the mouth, photophobia,dysphasia,recurrent otitismedia,chronic respiratory disease and dryness of the skin.

The following are less common:

Osteoporosis

Spontaneous fractures occuring in the long bones, neck of the femur and pelvis are well recognised in patients with Rheumatoid arthritis.

A small proportion of patients may develop Oseomalacia.

Lymphnode enlargement

Chronic positive RA or the neutropenic patient with felty's syndrome is particularly susceptible to infection.

It has been estimated that death from infections occurs at 8 to 10 times more the rate for the normal population.

Peripheral Oedema

Recurrent oedema of the lower limb is commonly found.

In some cases it develops around the acutely inflamed ankle joint.

STAGES OF RHEUMATOID ARTHIRITIS

The American College of Rheumatology has developed a system for classifying rheumatoid arthritis that is primarily based upon the X-ray appearance of the joints. This system classify the severity of Rheumatoid arthritis with respect to cartilage, ligaments, and bone.

Stage-I

- No damage seen on X-rays, although there may be signs of bone thinning

Stage-II

- On X-ray, evidence of bone thinning around a joint with or without slight bone damage
- Slight cartilage damage possible
- Joint mobility may be limited; no joint deformities observed
- Atrophy of adjacent muscle
- Abnormalities of soft tissue around joint possible

Stage-III

- On X-ray, evidence of cartilage and bone damage and bone thinning around the joint
- Joint deformity without permanent stiffening or fixation of the joint
- Extensive muscle atrophy
- Abnormalities of soft tissue around joint possible

Stage-IV

- On X-ray, evidence of cartilage and bone damage and osteoporosis around joint
- Joint deformity with permanent fixation of the joint (referred to as ankylosis)
- Extensive muscle atrophy
- Abnormalities of soft tissue around joint possible.

CRITERION:

JOINTS AFFECTED	**SCORE**
1 Large joint	0
2-10 Large joints	1
1-3 Small joints	2
4-10 Small joints	5
SEROLOGY	
Negative RF and ACPA	0

Low Positive RF or ACPA	2
High Positive RF or ACPA	3

DURATION OF SYMPTOMS

<6 weeks	0
>6 weeks	1

ACUTE PHASE REACTANTS

Normal CRP and ESR	0
Abnormal CRP or ESR	1

Patient with a score greater than or equal 6 are considered to have definite Rheumatiod Arthritis.

INVESTIGATIONS:

HAEMATOLOGICAL:

Total RBC Count

Haemoglobin Concentration

Total WBC count DC – Polymorphs

1. Lymphocytes
2. Eosinophils
3. Monocytes
4. Basophils

ESR

½ Hr

1 Hr

Blood Sugar

RBS

FBS

PPBS

Serum: Cholesterol.

Urea

Creatinine

Uric acid

URINE

Albumin

Sugar

Deposits

SPECIFIC INVESTIGATIONS

CRP

ASO Titre

Antinuclear antibody (ANA)

Rheumatoid factor (RF):

Designed to detect and measure the level of an antibody that acts against the blood component gamma globulin, this test is often positive in people with Rheumatoid arthritis.

Anti-cyclic citrullinated peptide (anti-CCP):

Also called anti-citrullinated protein antibodies (ACPA)

HLA tissue typing:

This test, which detects the presence of certain genetic markers in the blood, can often confirm a diagnosis of ankylosing spondylitis (a disease involving inflammation of the spine and sacroiliac joint) or reactive arthritis (a disease involving inflammation of the urethra, eyes and joints). The genetic marker HLA-B27 is almost always present in people with either of these diseases.

Lyme serology – This test detects an immune response to the infectious agent that causes Lyme disease and thus can be used to confirm a diagnosis of the disease.

Skin biopsy

Muscle biopsy

Joint fluid tests

RADIOLOGICAL INVESTIGATIONS:

The radiographic hallmarks of Rheumatoid arthritis are:

- **Soft tissue swelling:** Fusiform and Periarticular; it represents a combination of Joint Effusion, Oedema and Tenosynovitis
- **Osteoporosis:** initially juxta-articular, and later generalised.
- **Joint space narrowing:** Symmetrical or Concentric
- **Marginal Erosions:** Due to erosion by pannus of the bony “bare areas”

IN HANDS AND WRISTS:

There is a predilection for:

- PIP and MCP joints (especially 2nd and 3rd MCP)
- Ulnar –Styloid Process
- Triquetrum

As a rule, the DIP joints are spared.

Late changes include:

- Subchondral cyst formation: Destruction of cartilage presses synovial fluid into the bone
- Subluxation causing: Ulnar deviation of the MCP joints
- Boutonniere, Swan neck and Hitchhiker□s thumb deformities
- Carpal instability: Scapholunate dissociation, Ulnar translocation
- Ankylosis

FEET:

Similar to the hands, there is a predilection for the PIP and MTP joints (especially 4th and 5th MTP).

- Involvement of Subtalar joint
- Posterior Calcaneal tubercle erosion
- Hammer toe deformity
- Hallux valgus

SHOULDER:

- Erosion of the distal clavicle
- Marginal erosions of the Humeral head: The superolateral aspect is a typical location
- Reduction in the Acromio humeral distance: "High-riding shoulder" due to Sub acromial-Subdeltoid bursitis and high incidence of rotator cuff tear

HIP:

- Concentric loss of joint space where there is a tendency for superior loss of joint space
- Acetabular protrusion

KNEE:

- Joint effusion
- typically involves the lateral or non-weight bearing portion of the joint
- loss of joint space involving all three compartments
- lack of subchondral sclerosis and osteophytes, compared with OA
- Prepatellar bursitis

IN SPINE:

The Cervical spine is frequently involved in RA , whereas a Thoracic and Lumbar involvement are rare. Findings include:

- Erosion
- Atlanto axial subluxation-atlanto axial distance in more than 3 mm on a flexion radiograph Atlanto axial impaction (cranial settling).
- Erosion and fusion of uncovertebral (apophyseal joints) and facet joints
- Osteoporosis and osteoporotic fractures
- Erosion of spinous processes

Ultrasound:

Sonography can assess the soft tissue manifestations of RA,

- Synovial proliferation and inflammation of the superficial joints
- Tenosynovitis: Extensor carpi ulnaris tendon involvement is common in early disease and may lead to erosion of the ulnar styloid 2
- Bursitis

CT:

CT is not routinely used in the evaluation of peripheral RA. It has applications in imaging of the spine, and perioperative assessment.

MRI MRI is particularly sensitive to the early and subtle features of RA.

Features of RA best demonstrated with MRI include:

- Synovial hyperaemia: indication of acute inflammation
- Synovial hyperplasia (rice bodies)
- Pannus formation
- Decreased thickness of
- Subchondral cysts and erosions
- Juxta-articular bone marrow oedema
- Joint effusions

Differential diagnosis

The differential for the skeletal manifestations of RA includes:

• **Osteoarthritis involving the:** DIPs, PIPs, 1st CMC joints, Nonuniform joint space loss, subchondral sclerosis and osteophyte, soft tissue swelling, Heberdon□s node (DIPs) and Bouchard node (PIPs), no Erosions and no anklylosis.

• **Erosive osteoarthritis:** Clinically an acute inflammatory attacks (swelling, erythema, pain) in post menopausal woman typically includes the DIPs, PIPs 1st CMC joint 6, but not the metacarpo phalangeal (MCP) joints and large joints. Classic central erosions, possible ankylosis.

• **Psoriatic arthritis (PsA):** Commonly involves the hands and there is an interphalangeal predominant distribution in PsA. MCP joint pre dominance in rheumatoid arthritis (RA) starts with erosions in the margins and eventually involves the whole joint. Classic: “pencil in cup” and bone proliferation (unlike RA). Osteoporosis not a feature in PsA. MRI dynamic enhancement pattern may differentiate PsA from RA at 15 minutes

• **Reactive arthritis (Reiter’s syndrome):** Predilection for the lower limb Osteopenia and then Osteoporosis, uniform joint space loss, subchondral cyst formation, subluxations, marginal erosions but no bone formation, symmetrical involvement of the: PIPs, MCPs, and carpal bones.

• **Systemic lupus erythematosus (SLE)/Jaccoud arthropathy:** Joint space loss, subchondral sclerosis, osteophyte, and ulnar deviation of the phalanges without erosions

• **Calcium pyrophosphate dihydrate (CPPD) arthropathy:** usually only affects the MCP's: symmetric joint space narrowing, subchondral cysts, and osteophytes. unlike RA: chondrocalcinosis and no erosions.

• **Gout** usually in older men : Punched out erosions usually with a sclerotic border and overhanging edges, tophi, most commonly involves the 1st MTP known as podagra.

COMPLICATIONS:

Since rheumatoid arthritis is a systemic **scleritis** disease, its inflammation can affect organs and areas of the body other than the joints.

Inflammation of the glands of the eyes and mouth can cause dryness of these areas and is referred to as **Sjögren's syndrome.** Dryness of the eyes can lead to corneal abrasion.

Inflammation of the white parts of the eyes (the sclerae) is referred to as and can be very dangerous to the eye.

Rheumatoid inflammation of the lung lining **(pleuritis)** causes chest pain with deep breathing, shortness of breath, or coughing. The lung tissue itself can also become inflammed and scarred, and sometimes nodules of inflammation (rheumatoid nodules) develop within the lungs.

Inflammation of the tissue (pericardium) surrounding the heart, called **pericarditis**, can cause a chest pain that typically changes in intensity when lying down or leaning forward. Rheumatoid arthritis is associated with an increased risk for **heart attack.**

Rheumatoid disease can reduce the number of red blood cells **(anemia)** and white blood cells. Decreased white cells can be associated with an enlarged spleen referred to as **Felty's syndrome** and can increase the risk of infections.

The risk of lymph gland cancer **(lymphoma)** is higher in patients with rheumatoid arthritis, especially in those with sustained active joint inflammation.

Firm lumps or firm bumps under the skin, subcutaneous nodules called **rheumatoid nodules** can occur around the elbows and fingers where there is frequent pressure. Even though these nodules usually do not cause symptoms, occasionally they can become infected.

Caplan's syndrome (or) Rheumatoid pneumoconiosis still disease.

Nerves can become pinched in the wrists to cause **carpal tunnel syndrome.**

A rare, serious complication, usually with longstanding rheumatoid disease, is blood vessel inflammation **(vasculitis).** Vasculitis can impair blood supply to tissues and lead to tissue death **(NECROSIS)** leading to leg ulcers.

Factors which suggest poor prognosis

1. Young age
2. Female gender
3. positive family history
4. positive RA factor,Anti – CCP
5. Raised ESR
6. Extraarticular lesions
7. The presence of noduless

CHAPTER-IV
MATERIALS AND METHODS

The clinical study of "Vali Azhal Keel Vayu" is carried out in the Department of Pothu Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai under observation and guidance by the Head of the Department, Department of Pothu Maruthuvam.

Selection of the cases:

For this clinical study, 40 patients were selected according to the inclusion and exclusion criteria. 20 Out patients and remaining 20 In patients were treated in Out patient department and admitted in the In patient ward of the Government Siddha Medical College and Hospital, Palayamkottai.

Aetiological Factors:

The seasonal variations and precipitating factors like emotional stress, trauma, occupation and food habits were enquired and recorded. The socio economic status, family history and other significant diseases already treated, were thoroughly registered.

Inclusion criteria:

Age : 18-50 years

Sex : Both sex

Symmetrical joint involvement, painful small joints, florid morning stiffness more than hours per day, joint swelling especially in the interphalangeal joints, pain criteria score should be greater on equal to 6.

Exclusion criteria:

- Systemic Hypertension
- Diabetes mellitus
- Chronic Alcoholic and Smokers
- Pregnancy and lactating mothers
- Other locomotor disorders

- Pulmonary / Extra pulmonary Tuberculosis
- Psoriatic arthritis
- Gouty arthritis
- Chronic kidney disease
- Ischemic heart disease
- SLE

Diagnosis:

The diagnosis is made by following Siddha diagnostic methods:

- Poriyal Aridhal
- Pulanal Aridhal
- Vinnadhal
- Envagai Thervugal
- Udal Thathukal
- Kaalam
- Nilam

The following investigations were done in modern medicine aspects.

Haematological investigations:

- Total RBC Count
- HB%
- Total WBC count
- Differential count
- Erythrocyte sedimentation rate
- Blood sugar
- Serum Cholesterol
- Serum Urea
- Serum Creatinine
- Serum Uric acid

Urine analysis:

a. Albumin
b. Sugar
c. Deposits

Specific investigations:

a. CRP
b. RA factor
c. ASO titre

Radiological investigations (for selected cases):

X-ray of affected joints (AP and lateral view)

Assessment of results:

The results were assessed on the basis of symptomatic relief and **DISEASE ACTIVITY SCORE OF 28 JOINTS (DAS 28).**

The difference in the score before and after treatment represents the improvement in the treatment.

Treatment:

The clinical trial drug **"AKKINI CHOORANAM"** 4.2gm two times after food with sugar is given for 30 days till the end of the course. The Biochemical, Pharmacological and Acute toxicity studies were done in K.M.College of Pharmacy, Madurai, Tamilnadu.

All the patients admitted for the study were given uniformly regular hospital diet. After discharge all the patients were advised to attend the Out patient Department of Pothu Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai for further follow–up.

CHAPTER-V
RESULTS AND OBSERVATIONS

The results were observed regarding the following criteria by clinical trial study on 40 patients: Out of 40, 20 In patients and 20 Out patients of both sex.

The criteria were:

1. Sex Distribution
2. Age Distribution
3. Kaalam
4. Constitution of body
5. Gunam
6. Religion
7. Paruva Kaalam
8. Thinai
9. Socio – Economical status
10. Food habits
11. Family History
12. Occupation
13. Clinical Manifestation
14. Duration of Illness
15. Kanmenthiriyam
16. Kosam
17. Gnanendrium
18. Condition of Mukkutram
    a). Condition of Vatham
    b). Condition of Pitham
    c). Condition of Kapham
19. Involvement of Udal Kattugal (or) Udal thathukal
20. Conditions of Envagai Thervugal
21. Neer kuri
22. Nei kuri

23. Disease activity score
24. Assessment of outcome
25. Gradation of results
26. Laboratory Investigations
    a). Out patients
    b). In patients
27. Diseases activity pain score
    a). Out patients
    b). In patients
28. Case summary
    a). Out patients
    b). In patients

1. **SEX DISTRIBUTION:**

Table-1 Illustrates the Distribution of Sex and its percentage.

TABLE-1

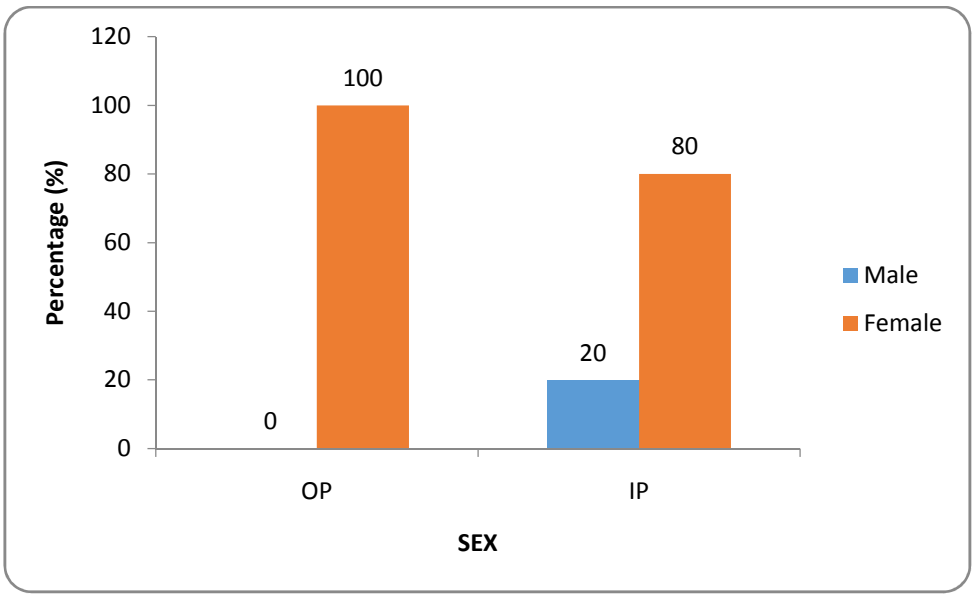
DISTRIBUTION OF SEX

Sl. No.	Sex	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Male	-	-	4	20%
2.	Female	20	100%	16	80%
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients 0% were Male and 100% were Female. Among 20 In patients, 20% were Male and 80% were Female.

FIGURE-1

DISTRIBUTION OF SEX



2. AGE DISTRIBUTION:

Table-2 Illustrates the Distribution of Age and its percentage.

TABLE-2

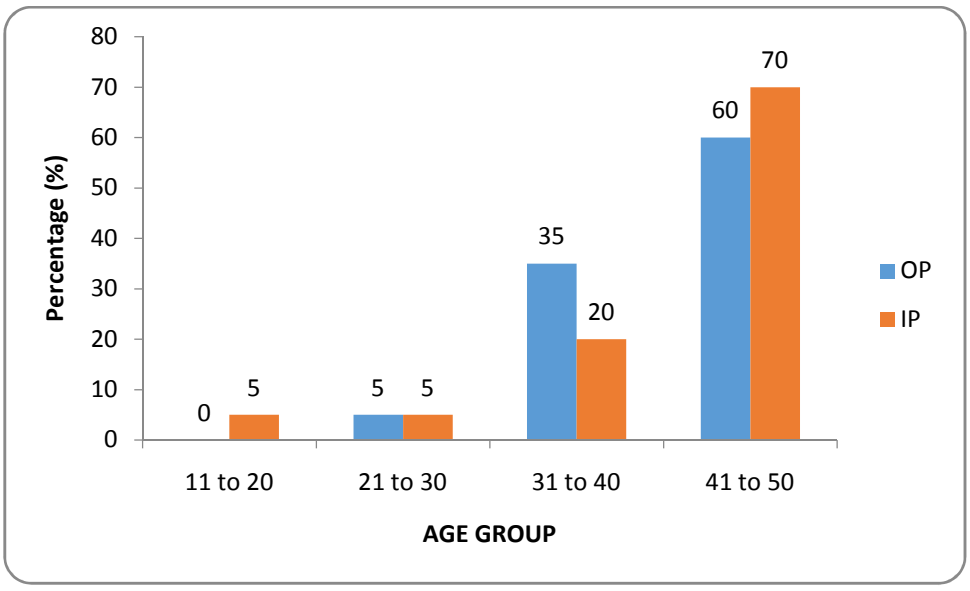
DISTRIBUTION OF AGE

Sl. No.	Age group in years	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	11 to 20	-	-	1	5%
2.	21 to 30	1	5%	1	5%
3.	31 to 40	7	35%	4	20%
4.	41 to 50	12	60%	14	70%
	Total	**20**	**100%**	**20**	**100%**

From the above table it is observed that the highest incidence of Vali Azhal Keel Vayu in Out patients is among the age group of 41 to 50 with 60% and 31 to 40 with 35%, 5% were in the age group of 21 to 30 years. Among 20 In patients 5% were in the age group of 11 to 20 years and 21 to 30 years , 20% were in the age group of 31 to 40 years , 70% with the highest incidence in the age group of 41 to 50 years.

FIGURE-2

DISTRIBUTION OF AGE



3. **KAALAM DISTRIBUTION:**

Table-3 Illustrates the Distribution of Kaalam and its percentage.

TABLE-3

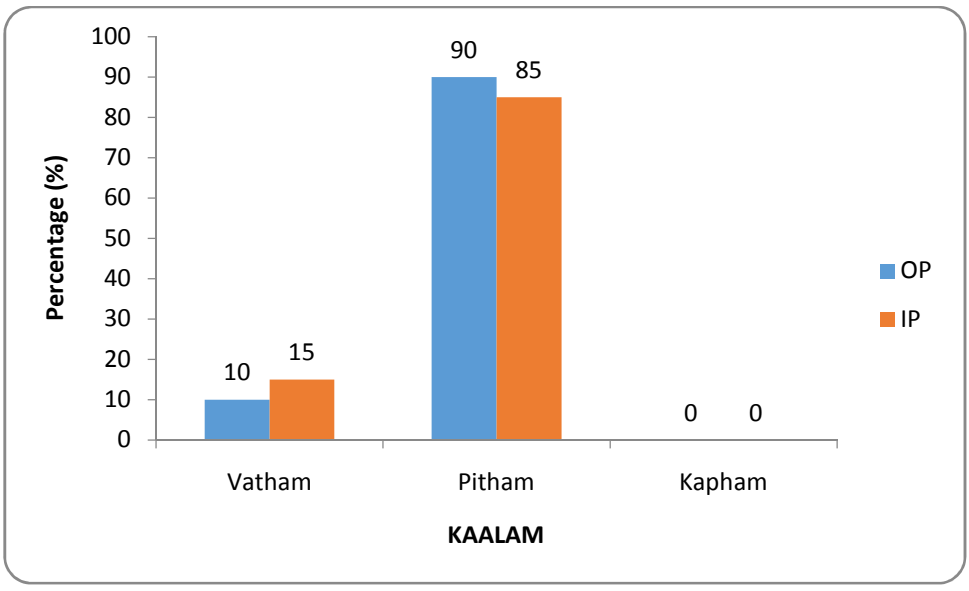
DISTRIBUTION OF KAALAM

Sl. No.	Kaalam	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Vatham	2	10%	3	15%
2.	Pitham	18	90%	17	85%
3.	Kapham	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, 90% were affected in Pitha Kaalam and 10% were affected in VathaKaalam. Among 20 In patients, 85% were affected in Pitha Kaalam and 15% were affected in Vatha Kaalam.

FIGURE-3

DISTRIBUTION OF KAALAM



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4 CONSTITUTION OF BODY:

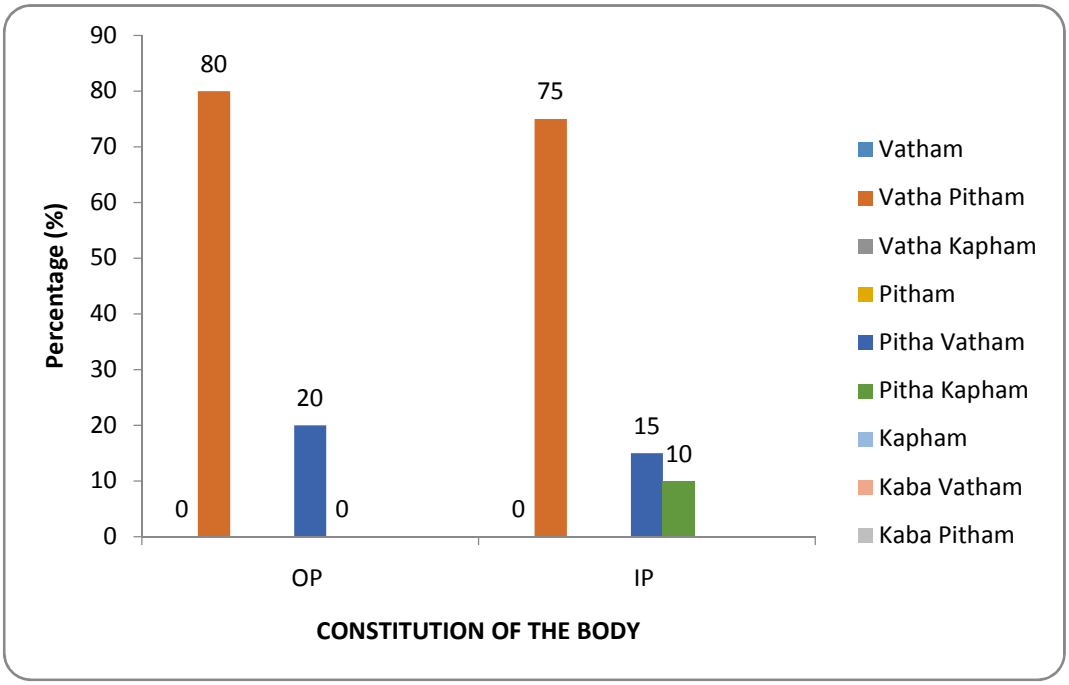
Table-4 Illustrates the Distribution of Constitution Body and its percentage.

TABLE-4
DISTRIBUTION OF CONSTITUTION OF BODY

Sl. No.	Constitution of body	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Vatham	-	-	-	-
2.	Vatha Pitham	16	80%	15	75%
3.	Vatha Kapham	-	-	-	-
4.	Pitham	-	-	-	-
5.	Pitha Vatham	4	20%	3	15%
6.	Pitha Kapham	-	-	2	10%
7.	Kapham	-	-	-	-
8.	Kaba Vatham	-	-	-	-
9.	Kaba Pitham	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

Vatha Pitha Thegi register high incidence of Vali Azhal Keel Vayu with 80% OP and 75% IP. Remaining Pitha Vatha Thegi of 20% OP and 15% IP, Pitha Kapham Thegi of 10% In patients.

FIGURE-4 DISTRIBUTION OF CONSTITUTION OF BODY



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5.GUNAM:

Table-5 Illustrates the Distribution of Gunam and its percentage.

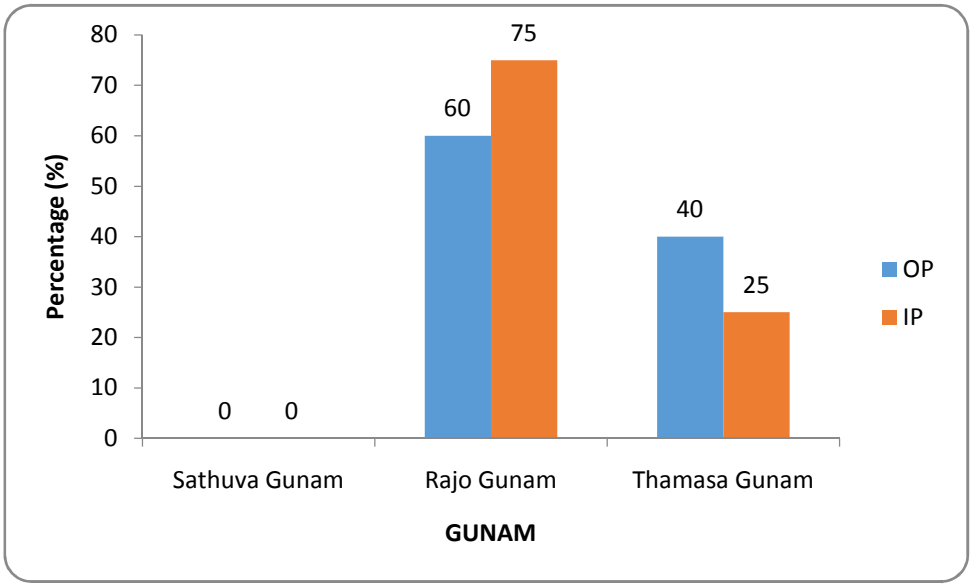
TABLE-5 DISTRIBUTION OF GUNAM

Sl. No.	Gunam	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Sathuva Gunam	-	-	-	-
2.	Rajo Gunam	12	60%	15	75%
3.	Thamasa Gunam	8	40%	5	25%
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, 60% were Rajo gunam and 40% were Thamasa gunam.

Among 20 In patients, 75% were Rajo gunam and 25% were Thamasa gunam.

FIGURE-5 DISTRIBUTION OF GUNAM



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6. RELIGION:

Table-6 Illustrates the Distribution of Religion and its percentage.

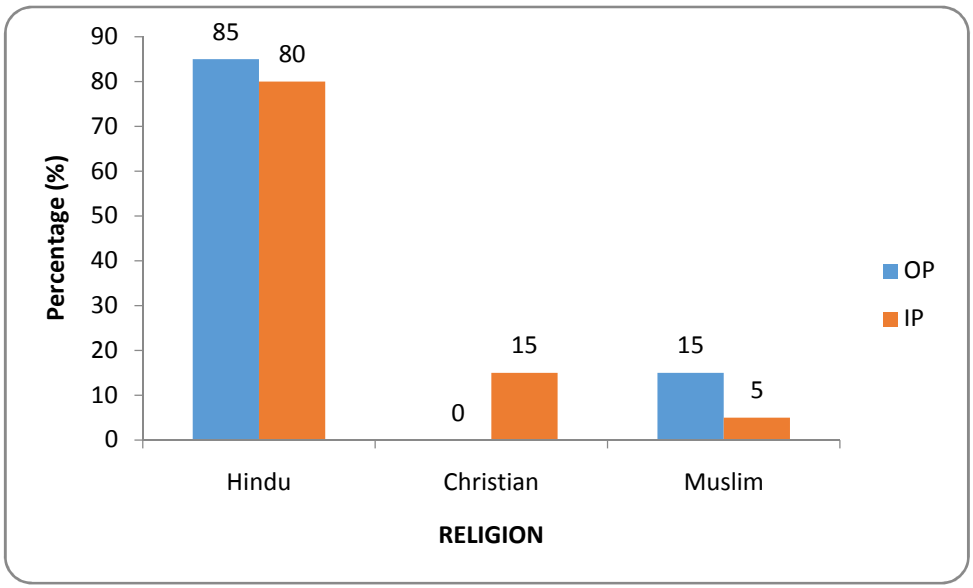
TABLE-6

DISTRIBUTION OF RELIGION

Sl. No.	Religion	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Hindu	17	85%	16	80%
2.	Christian	-	-	3	15%
3.	Muslim	3	15%	1	5%
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, 85% were Hindus, 15% were Muslims. Among 20 In patients, 80% were Hindus,15% were Christians and 5% were Muslims .

FIGURE-6 DISTRIBUTION OF RELIGION



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7. PARUVA KAALAM:

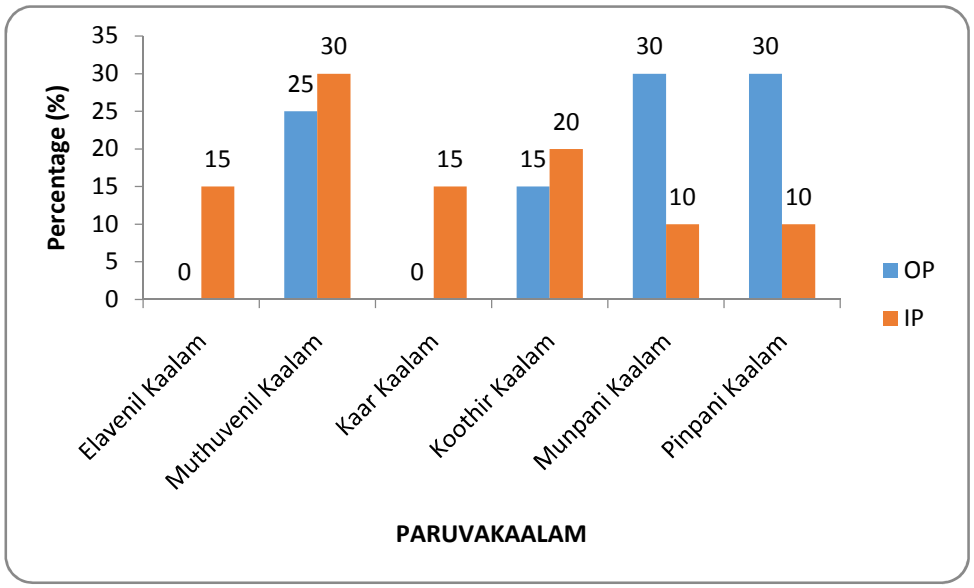
Table-7 Illustrates the Distribution of Paruva Kaalam and its percentage.

TABLE-7 DISTRIBUTION OF PARUVA KAALAM

Sl. No.	Paruva Kaalam	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Elavenil Kaalam	-	-	3	15%
2.	Muthuvenil Kaalam	5	25%	6	30%
3.	Kaar Kaalam	-	-	3	15%
4.	Koothir Kaalam	3	15%	4	20%
5.	Munpani Kaalam	6	30%	2	10%
6.	Pinpani Kaalam	6	30%	2	10%

Among 20 Out patients, 30% of cases were in Munpani Kaalam and Pinpani Kaalam,25% of cases were in Muthuvenil Kaalam and 15% of cases were in Koothir Kaalam. Among 20 In patients, 30% of cases were in Muthuvenil Kaalam, 20% of cases were in Koothir Kaalam and 15% of cases were in Elavenil Kaalam and Kaar Kaalam,10% of cases were in Munpani and Pinpani Kaalam.

FIGURE-7 DISTRIBUTION OF PARUVA KAALAM



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8. THINAI:

Table-8 Illustrates the Distribution of Thinai and its percentage.

TABLE-8

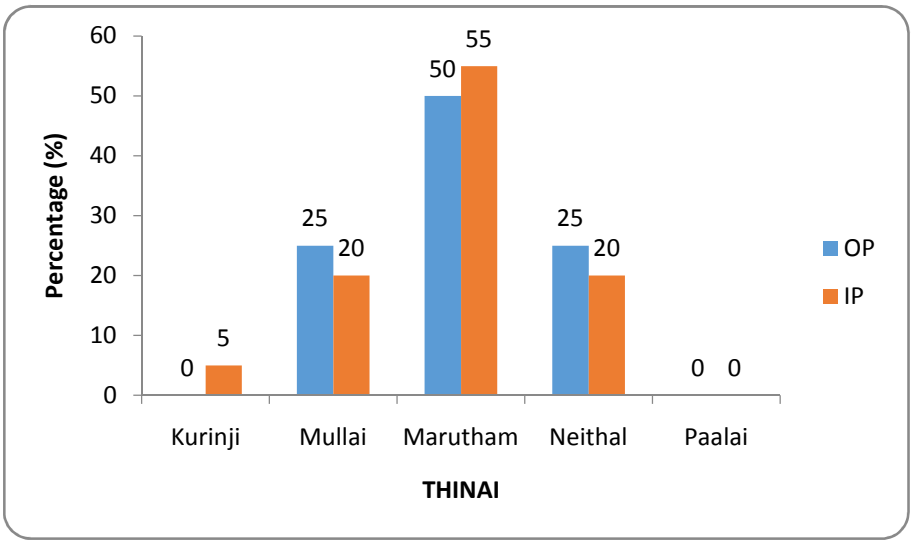
DISTRIBUTION OF THINAI

Sl. No.	Thinai	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Kurinji	-	-	1	5%
2.	Mullai	5	25%	4	20%
3.	Marutham	10	50%	11	55%
4.	Neithal	5	25%	4	20%
5.	Paalai	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, 50% were in Marutham and 25% were in Neithal and Mullai.

Among 20 In patients, 55% were in Marutham and 20% were in Neithal and Mullai, 5% were in Kurinji

FIGURE-8 DISTRIBUTION OF THINAI



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9. SOCIO - ECONOMICAL STATUS:

Table-9 Illustrates the Socio – Economical Status of patients and its percentage.

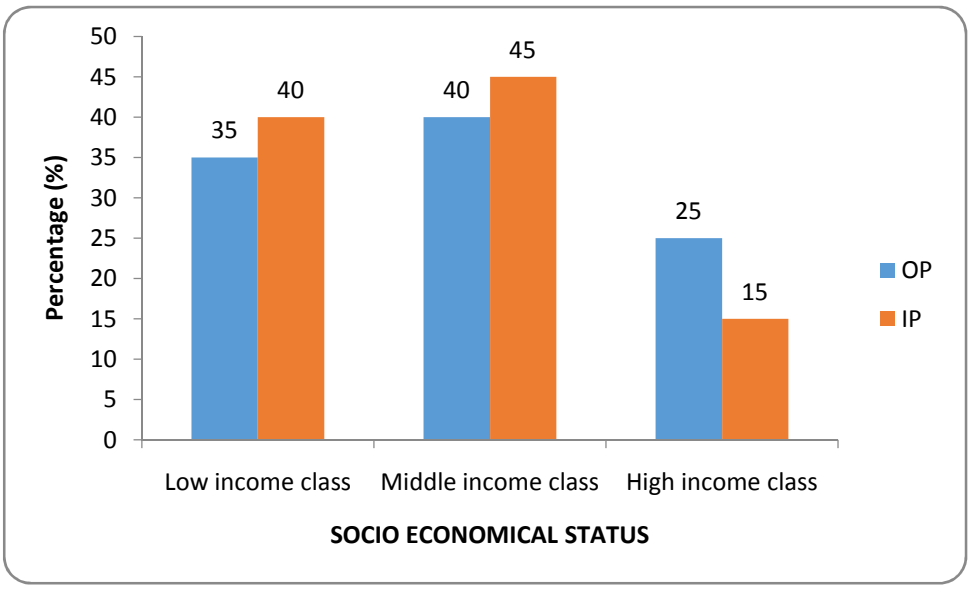
TABLE-9

SOCIO – ECONOMICAL STATUS

Sl. No.	Socio – Economical Status	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Low income class	7	35%	8	40%
2.	Middle income class	8	40%	9	45%
3.	High income class	5	25%	3	15%
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, 35% were in Low income class, 40% were in Middle income class and 25% were in High income class. Among 20 In patients, 40% were in Low income class, 45% were in Middle income class and 15% were in High income class.

FIGURE-9 SOCIO – ECONOMICAL STATUS



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10 FOOD HABITS:

Table-10 Illustrates the Distribution of diet among the patients and its percentage.

TABLE-10

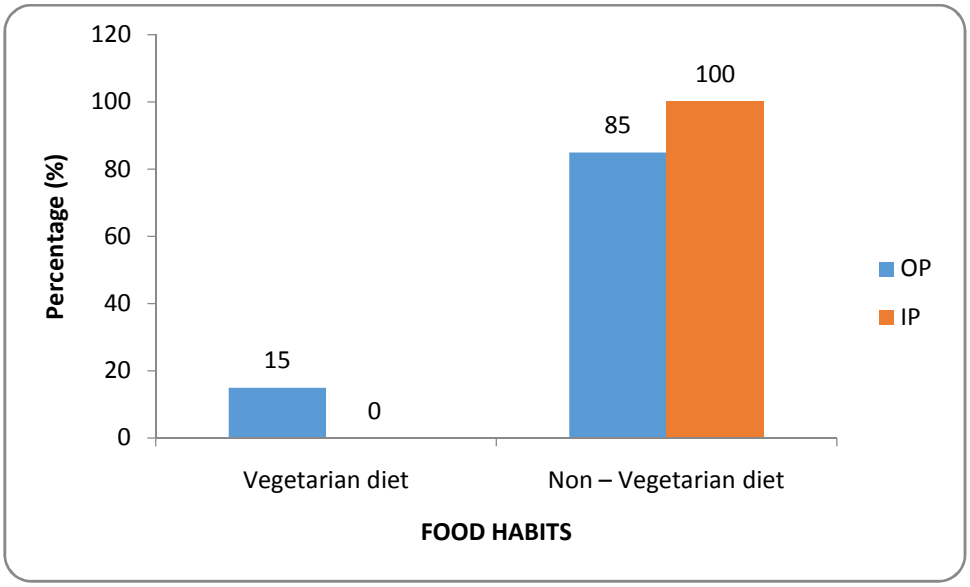
FOOD HABITS

Sl. No.	Food Habits	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Vegetarian diet	3	15%	-	-
2.	Non – Vegetarian diet	17	85%	20	100%
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, 15% were Vegetarian and 85% were non – vegetarian.

Among 20 In patients, 0% were vegetarian and 100% were non – vegetarian.

FIGURE-10 FOOD HABITS



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11. FAMILY HISTORY:

Table-11 Illustrates the Family History and its percentage.

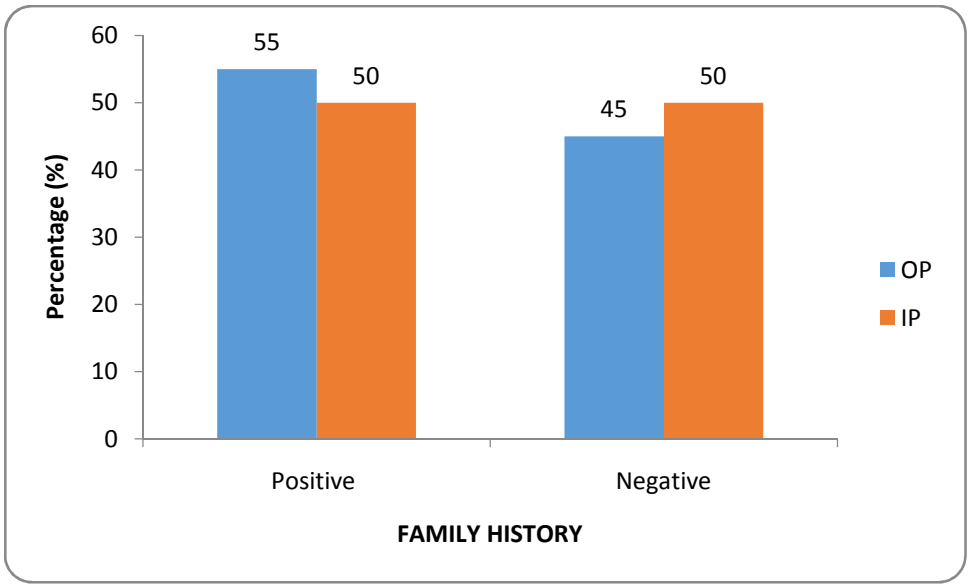
TABLE-11

FAMILY HISTORY

Sl. No.	Family History	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Positive	11	55%	10	50%
2.	Negative	9	45%	10	50%
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, 55% have positive Family History and 45% don't have any positive Family History. Among 20 In patients, 50% have positive Family History and 50% don't have any positive Family History.

FIGURE-11 FAMILY HISTORY



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12 OCCUPATION:

Table-12 Illustrates the Occupation and its percentage.

TABLE-12

OCCUPATION

Sl. No.	Occupation	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Agricultural labours	2	10%	4	20%
2.	Coolie	3	15%	5	25%
3.	House wife	11	55%	4	20%
4.	Tailor	2	10%	2	10%
5.	Loadman	-	-	1	5%
6.	Driver	-	-	1	5%
7.	Beedi Worker	1	5%	-	-
8.	Business	-	-	1	5%
9.	Sales Women	1	5%	-	-
10.	Flower Maker	-	-	1	5%
11.	Mason	-	-	1	5%
	Total	**20**	**100%**	**20**	**100%**

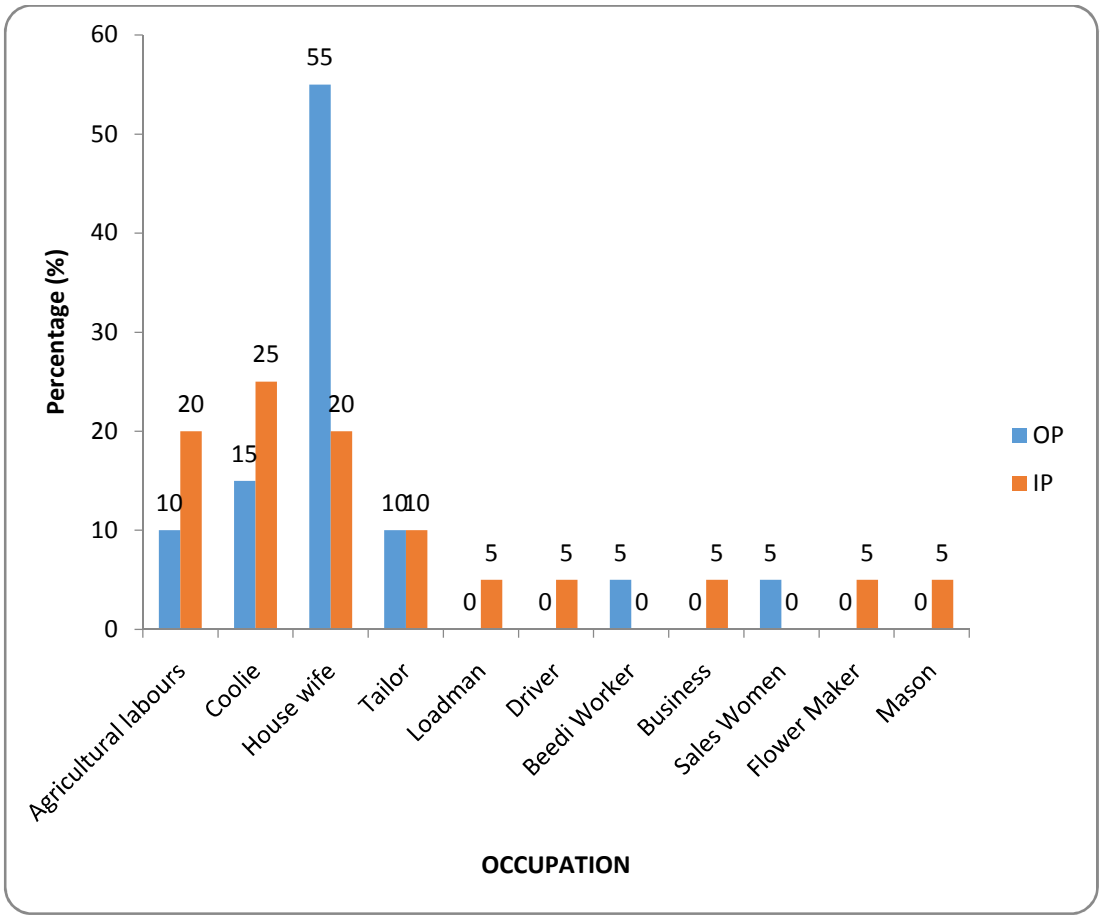
Among 20 Out patients, 10% Agricultural labours, 15% Coolie, 55% House Wife, 10% Tailor, 5% Beedi worker and Sales women.

Among 20 In patients, 20% Agricultural labours, 25% Coolie, 20% House Wife, 5% Loadman, 5% Driver,5% Business,5% Flower Maker,5% Mason, 10% Tailor were observed.

.

FIGURE-12

OCCUPATION



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13 CLINICAL MANIFESTATION:

Table-13 Illustrates the Clinical Manifestation and its percentage.

TABLE-13

CLINICAL MANIFESTATION

Sl. No.	Symptoms	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Arthritis involving more than 3 joints	20	100%	20	100%
2.	Severe pain and swelling	20	100%	20	100%
3.	Symmetrical joint involvement	20	100%	20	100%
4.	Morning stiffness	17	85%	10	50%
5.	Anorexia	4	20%	7	35%
6.	Rheumatoid nodules	3	15%	5	25%
7.	Depression	9	45%	11	55%

Among 20 Out patients, 100% cases have arthritis involving more than 3 joints, severe pain and swelling, symmetrical joint involvement,85% have morning stiffness , 45% have depression, 20% have Anorexia, 15% have Rheumatoid nodules. Among 20 In patients, 100% cases have arthritis involving more than 3 joints, severe pain and swelling, symmetrical joint involvement,50% have morning stiffness, 55%% have depression, 25% have rheumatoid nodules, 35% have anorexia.

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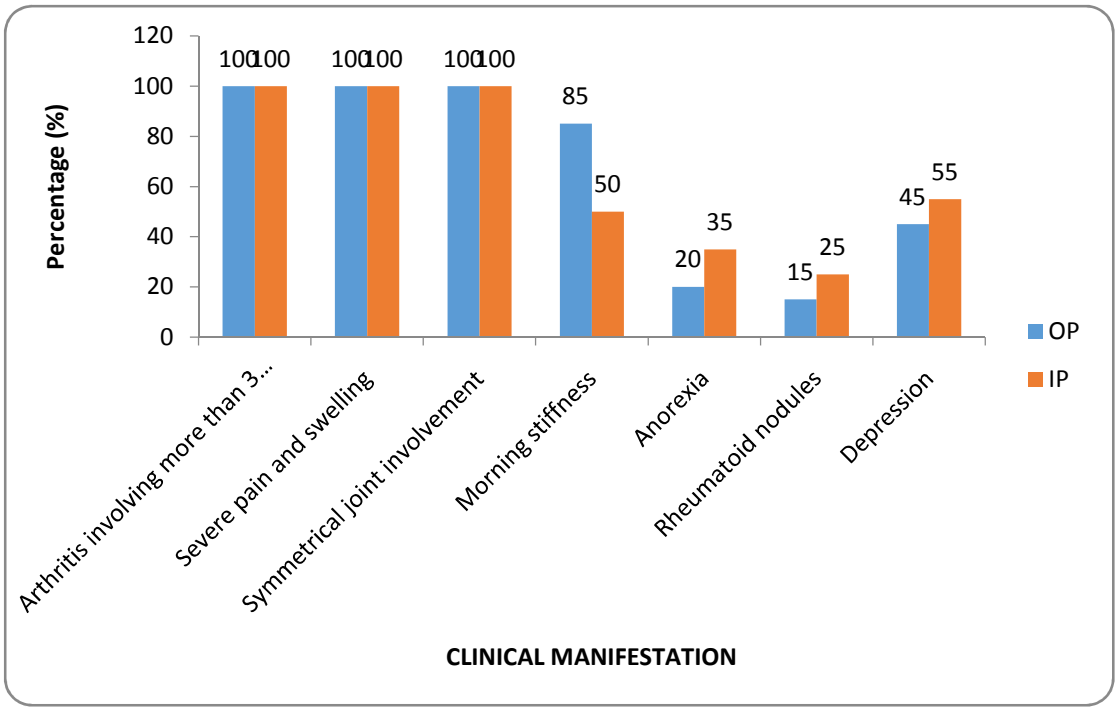
FIGURE-13

CLINICAL MANIFESTATION



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14 DURATION OF ILLNESS:

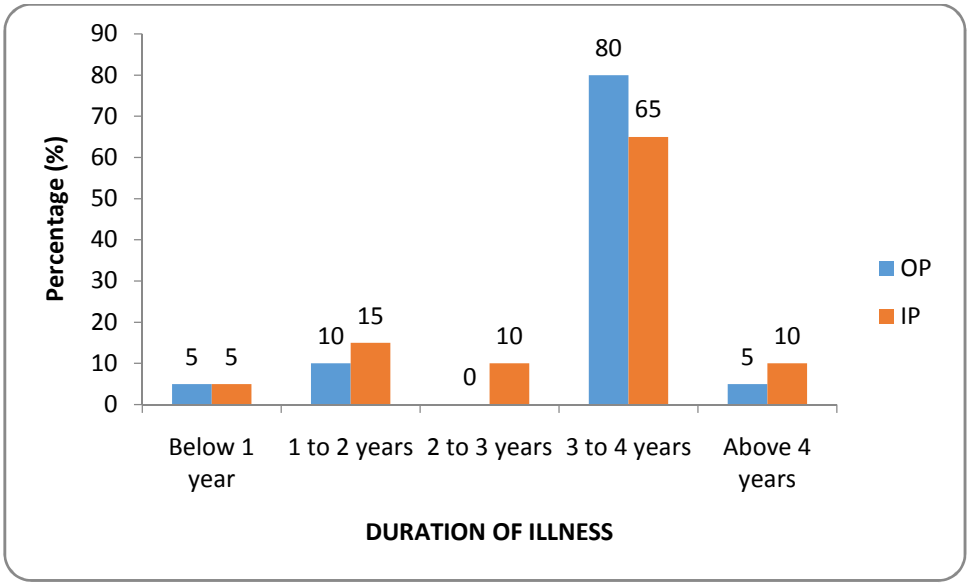
Table-14 Illustrates the Duration of Illness and its percentage.

TABLE-14 DURATION OF ILLNESS

Sl. No.	Duration of illness	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Below 1 year	1	5%	1	5%
2.	1 to 2 years	2	10%	3	15%
3.	2 to 3 years	-	-	2	10%
4.	3 to 4 years	16	80%	13	65%
5.	Above 4 years	1	5%	2	10%
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, Duration of Illness was 5% below 1 year, 10% in 1 to 2 years,80% in 3 to 4 years and 5% in above 4 years. Among 20 In patients, Duration of Illness was 5% below 1 year, 15% in 1 to 2 years, 10% in 2 to 3 years and 65% in 3 to 4 years,10% above 4 year.

FIGURE-14 DURATION OF ILLNESS



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15 KANMENTHIRIYAM:

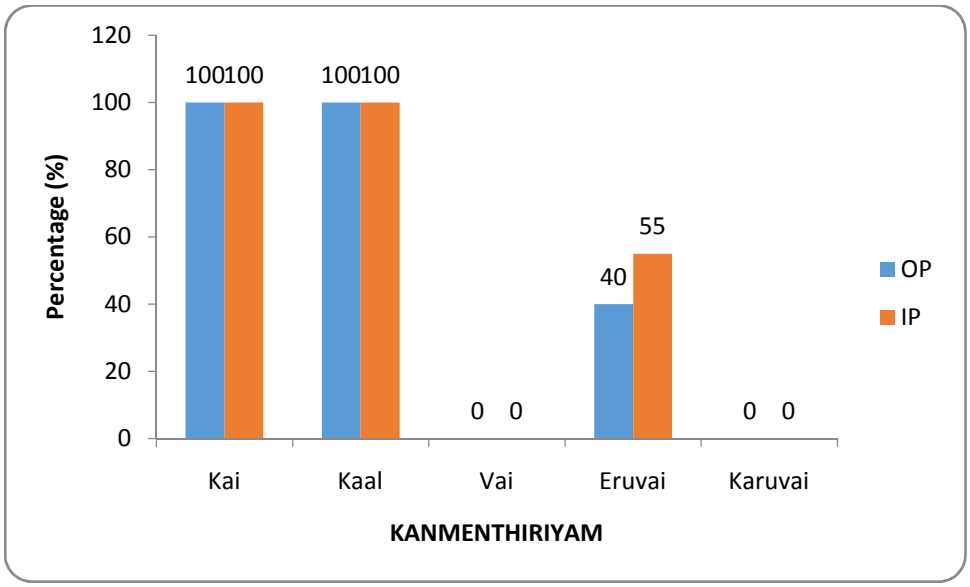
Table-15 Illustrates the Kanmenthiriyam and its percentage.

TABLE-15 KANMENTHIRIYAM

Sl. No.	Kanmenthiriyam	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Kai	20	100%	20	100%
2.	Kaal	20	100%	20	100%
3.	Vai	-	-	-	-
4.	Eruvai	8	40%	11	55%
5.	Karuvai	-	-	-	-

Among 20 Out patients, 100% cases were affected in Kai, Kaal, 40% cases were affected in Eruvai. Among 20 In patients, 100% cases were affected in Kai, Kaal, 55% cases were affected in Eruvai.

FIGURE-15 KANMENTHIRIYAM



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16 KOSAM

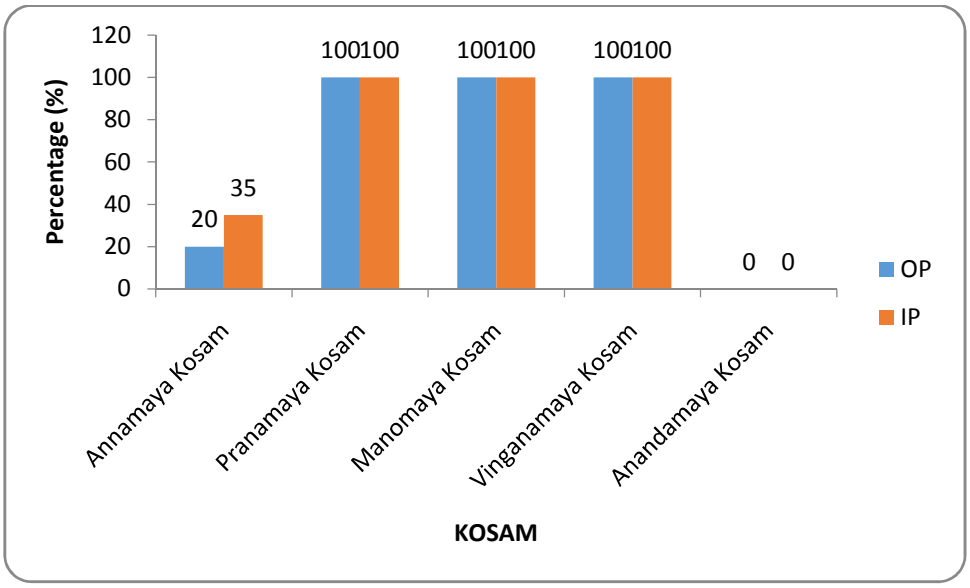
Table-16 Illustrates the Kosam and its percentage.

TABLE-16 KOSAM

Sl. No.	Kosam	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Annamaya Kosam	4	20%	7	35%
2.	Pranamaya Kosam	20	100%	20	100%
3.	Manomaya Kosam	20	100%	20	100%
4.	Vinganamaya Kosam	20	100%	20	100%
5.	Anandamaya Kosam	-	-	-	-

Among 20 Out patients, 20% were affected in Annamya Kosam, 100% were affected in Pranamaya Kosam,Manomya Kosam and Vinganamaya Kosam. Among 20 In patients, 35% were affected in Annamya Kosam and 100% were affected in Pranamaya Kosam,Manomya Kosam and Vinganamaya Kosam.

FIGURE-16 KOSAM



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17 GNANENDRIUM:

Table-17 Illustrates the Gnanendrium and its percentage.

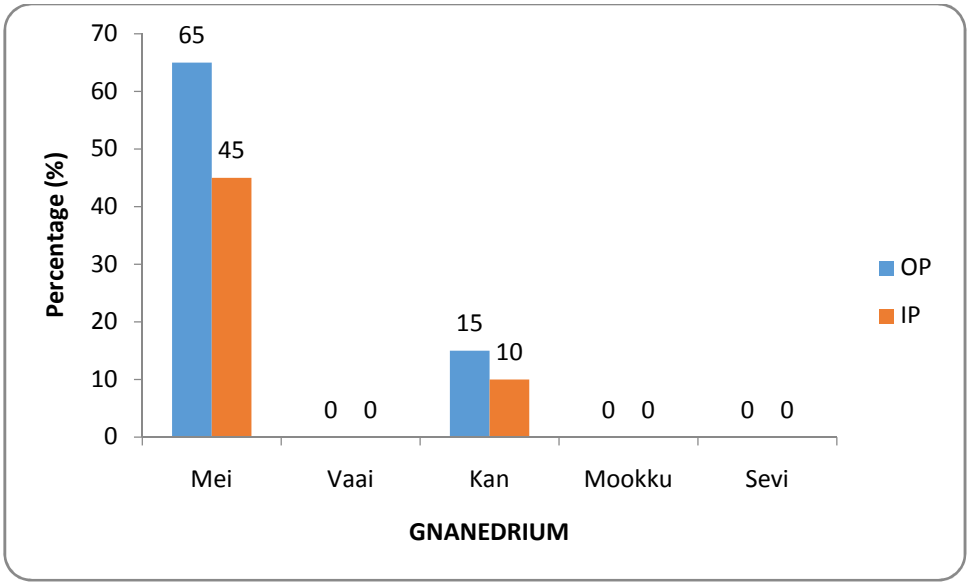
TABLE-17 GNANENDRIUM

Sl. No.	Gnanendrium	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Mei	13	65%	9	45%
2.	Vaai	-	-	-	-
3.	Kan	3	15%	2	10%
4.	Mookku	-	-	-	-
5.	Sevi	-	-	-	-

Among 20 Out patients, 65% of the patients were affected in Mei, 15% of the patients were affected in kan.

Among 20 In patients, 45% of the patients were affected in Mei, 10% of the patients were affected in kan.

FIGURE-17 GNANENDRIUM



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18 CONDITION OF MUKKUTRAM:

18 (a) Condition of Vatham.

Table-18 Illustrates the Condition of Vatham and its percentage.

TABLE-18 (a)

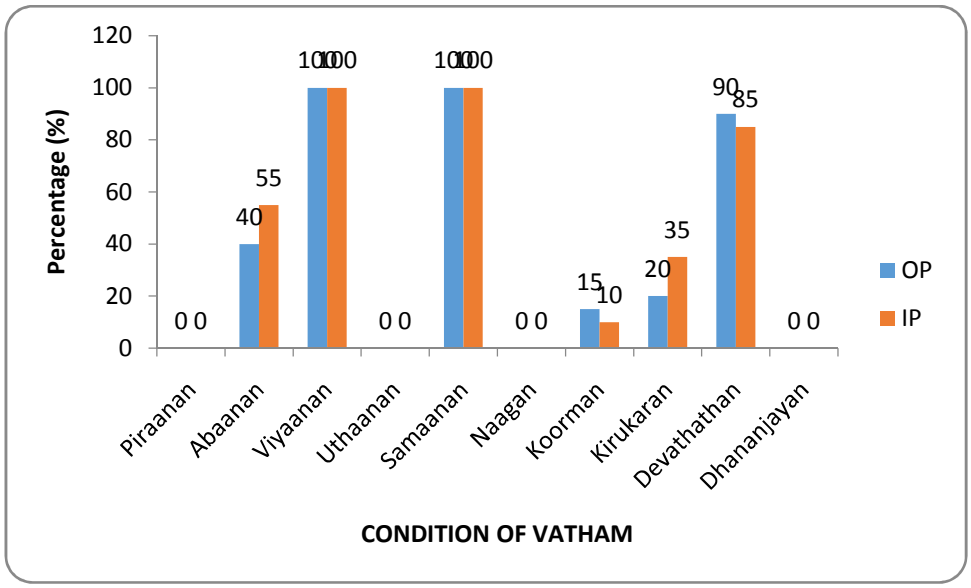
CONDITION OF VATHAM

Sl. No.	Condition of Vatham	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Piraanan	-	-	-	-
2.	Abaanan	8	40%	11	55%
3.	Viyaanan	20	100%	20	100%
4.	Uthaanan	-	-	-	-
5.	Samaanan	20	100%	20	100%
6.	Naagan	-	-	-	-
7.	Koorman	3	15%	2	10%
8.	Kirukaran	4	20%	7	35%
9.	Devathathan	18	90%	17	85%
10.	Dhananjayan	-	-	-	-

Viyaanan, Samaanan, were affected in 100% of both Out patients and In patients. Abaanan was affected in 40% of Out patients, and 55% of the In patients, Kirugaran was affected in 20% of Out Patients, and 35% of In patients. Devathathan was affected in 90% of Out patients, and 85% of In patients. Koorman was affected in 15% of Out patients and 10% of In patients,

FIGURE-18 (a)

CONDITION OF VATHAM



18 (b). CONDITION OF PITHAM

Table-18 (b) Illustrates the Condition of Pitham and its percentage.

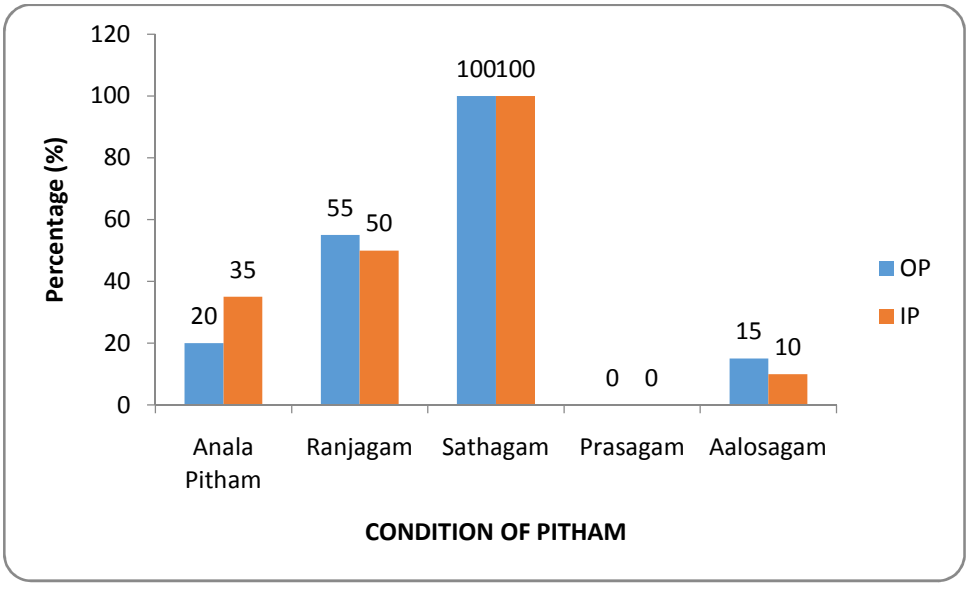
TABLE-18 (b)

CONDITION OF PITHAM

Sl. No.	Pitham	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Anala Pitham	4	20%	7	35%
2.	Ranjagam	11	55%	10	50%
3.	Sathagam	20	100%	20	100%
4.	Prasagam	-	-	-	-
5.	Aalosagam	3	15%	2	10%

Among 20 Out patients, 20% affected in Anala Pitham, 55% in Ranjaga Pitham and 100% in Sathaga Pitham, 15% in Aalosagapitham. Among 20 In patients, 35% affected in Anala Pitham, 50% in Ranjaga Pitham and 100% in Sathaga Pitham, 10% in Aalosagapitham.

FIGURE-18 (b) CONDITION OF PITHAM



18 (c) CONDITION OF KAPHAM

Table-18 (c) Illustrates the Condition of Kapham and its percentage.

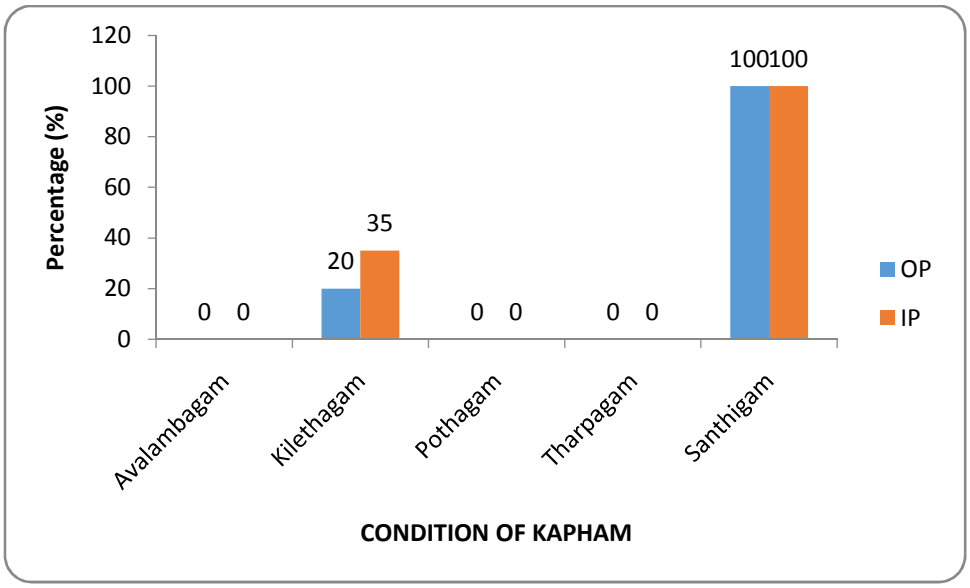
TABLE-18 (c)

CONDITION OF KAPHAM

Sl. No.	Kapham	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Avalambagam	-	-	-	-
2.	Kilethagam	4	20%	7	35%
3.	Pothagam	-	-	-	-
4.	Tharpagam	-	-	-	-
5.	Santhigam	20	100%	20	100%

Kilethagam was affected in 20% of Out patients and 35% of In patients. Santhigam was affected in 100% of both Out patients and In patients.

FIGURE-18 (c) CONDITION OF KAPHAM



19. INVOLVEMENT OF UDAL KATTUGAL (OR) UDAL THATHUKAL

Table-19 Illustrates the involvement of Udal Kattugal (or) Udal Thathukal and its percentage.

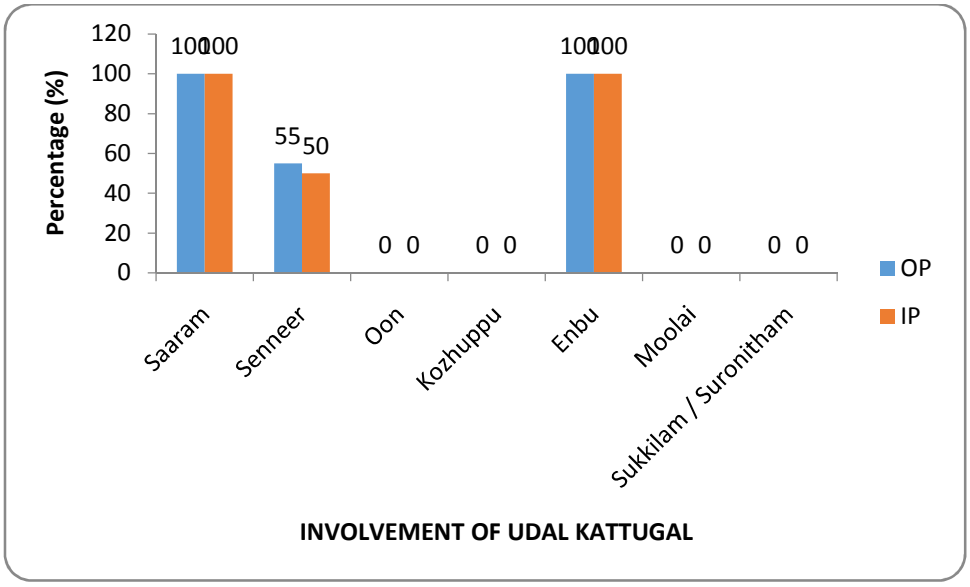
TABLE-19

INVOLVEMENT OF UDAL KATTUGAL (OR) UDAL THATHUKAL

Sl. No.	Udal Kattugal	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Saaram	20	100%	20	100%
2.	Senneer	11	55%	10	50%
3.	Oon	-	-	-	-
4.	Kozhuppu	-	-	-	-
5.	Enbu	20	100%	20	100%
6.	Moolai	-	-	-	-
7.	Sukkilam / Suronitham	-	-	-	-

Among 20 Out patients and In patients Saaram, Enbu were affected in 100% of the cases. senneer was affected in 55% of OP and 50% of IP.

FIGURE-19 INVOLVEMENT OF UDAL KATTUGAL



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20 CONDITIONS OF ENVAGAI THERVUGAL

Table-20 Illustrates the conditions of Envagai Thervugal and its percentage.

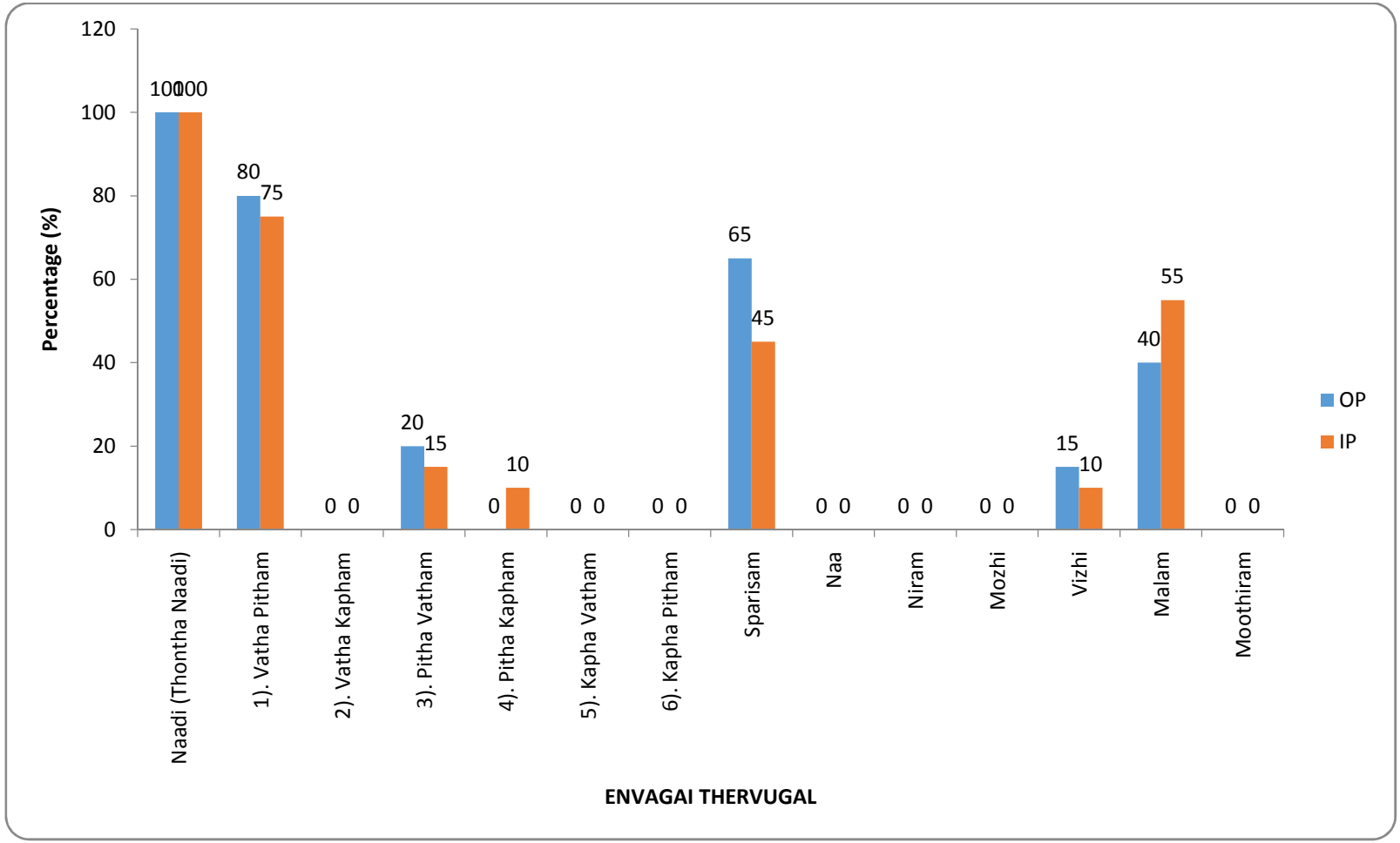
TABLE-20

CONDITION OF ENVAGAI THERVUGAL

Sl. No.	Envagai Thervugal	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Naadi (Thontha Naadi)	20	100%	20	100%
	1). Vatha Pitham	16	80%	15	75%
	2). Vatha Kapham	-	-	-	-
	3). Pitha Vatham	4	20%	3	15%
	4). Pitha Kapham	-	-	2	10%
	5). Kapha Vatham	-	-	-	-
	6). Kapha Pitham	-	-	-	-
2.	Sparisam	13	65%	9	45%
3.	Naa	-	-	-	-
4.	Niram	-	-	-	-
5.	Mozhi	-	-	-	-
6.	Vizhi	3	15%	2	10%
7.	Malam	8	40%	11	55%
8.	Moothiram	-	-	-	-

Among 20 Out patients,15 % were affected in Vizhi, 40% were affected in Malam, 65% was affected in Sparisam, Naadi-80% with Vatha Pitham, 20% with Pitha Vatham . Among 20 In patients, 10% were affected in Vizhi, 55% was affected in Malam, 45% was affected in Sparisam, Naadi-75% with Vatha Pitham ,15% with Pitha Vatham and 10% with Pitha Kapham.

FIGURE-20 CONDITION OF ENVAGAI THERVUGAL



21 NEER KURI

Table-21 Illustrates the Neer kuri condition and its percentage.

TABLE-21

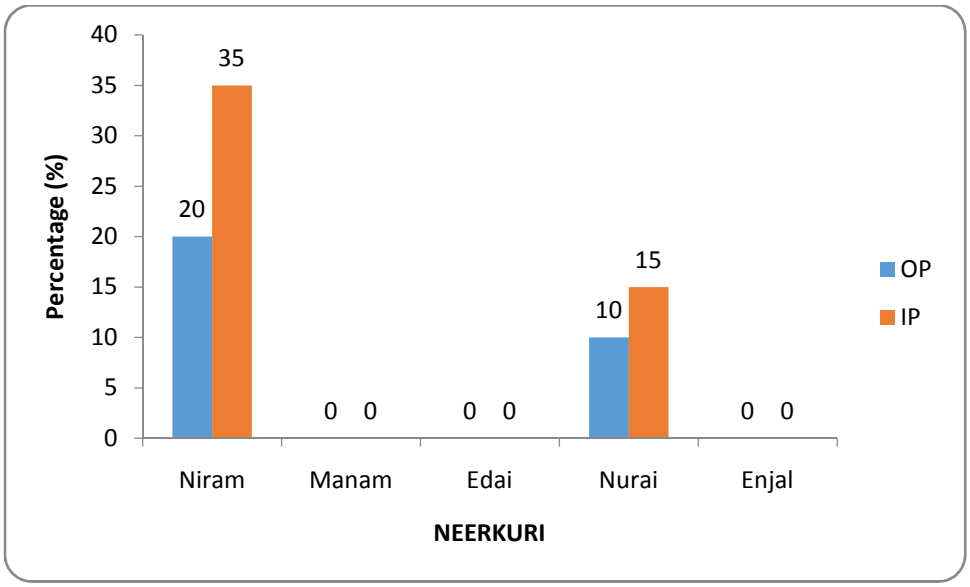
NEER KURI

Sl. No.	Neer kuri	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Niram	4	20%	7	35%
2.	Manam	-	-	-	-
3.	Edai	-	-	-	-
4.	Nurai	2	10%	3	15%
5.	Enjal	-	-	-	-

Niram was ffected in 20% of Out patients and 35% of In patients. Nurai was affected in 10% of Out patients and 15% of In patients.

FIGURE-21

NEER KURI



22. NEI KURI

Table-22 Illustrates the Nei Kuri conditions and its percentage.

TABLE-22

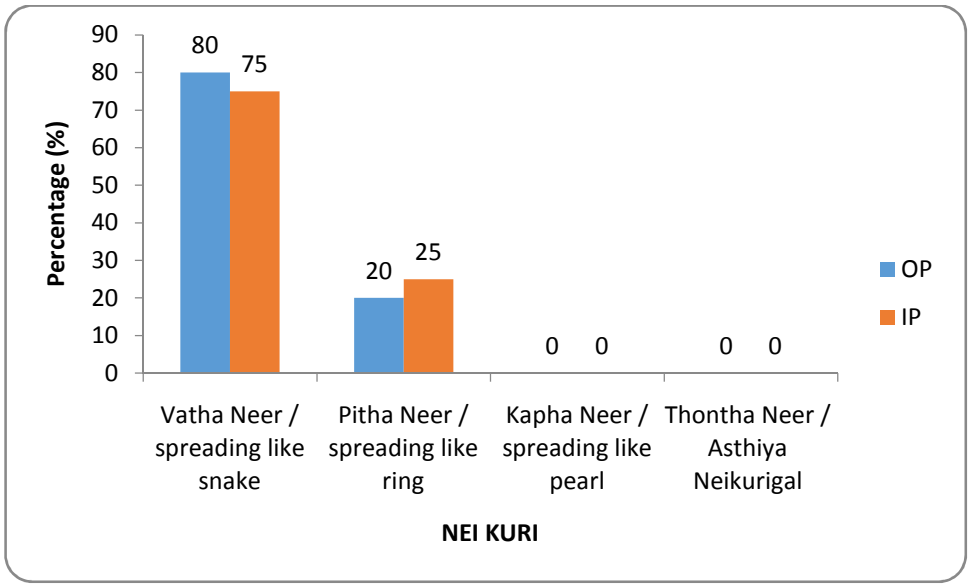
NEI KURI

Sl. No.	Nei Kuri	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Vatha Neer / spreading like snake	16	80%	15	75%
2.	Pitha Neer / spreading like ring	4	20%	5	25%
3.	Kapha Neer / spreading like pearl	-	-	-	-
4.	Thontha Neer / Asthiya Neikurigal	-	-	-	-

Among 20 Out patients, 80% had Vatha Neer, 20% had Pitha neer.

Among 20 In patients, 75% had Vatha Neer, 25% had Pitha Neer.

FIGURE-22 NEI KURI



23. DISEASES ACTIVITY SCORE

Table-23 Illustrates the Diseases activity score.

TABLE-23

DISEASES ACTIVITY SCORE

Sl. No.	Disease activity score	Before Treatment				After Treatment			
		Out Patients (OP)		In Patients (IP)		Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)	No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Low DAS Score (Tenderness)	20	100%	20	100%	10	50%	12	60%
2.	Moderate DAS score (Swelling)	20	100%	20	100%	6	30%	4	20%
3.	High DAS score (VAS)	20	100%	20	100%	4	20%	4	20%

Lower disease activity $2.6 < DAS28 \leq 3.2$.

Moderate disease activity $3.2 < DAS28 \leq 5.1$.

High disease activity $DAS28 > 5.1$.

Reference:

Prevoo ML, Van't Hof MA Kuper HH, Van Leeuwen MA, Van de Putte LB, Van Rie / PL (1995). "Modified disease activity scores that include twenty-eightjoints counts. Development and validation in a prospective longitudinal study of patients with rheumatoid arthritis". Arthritis Rheum. 38 (1):44-8.

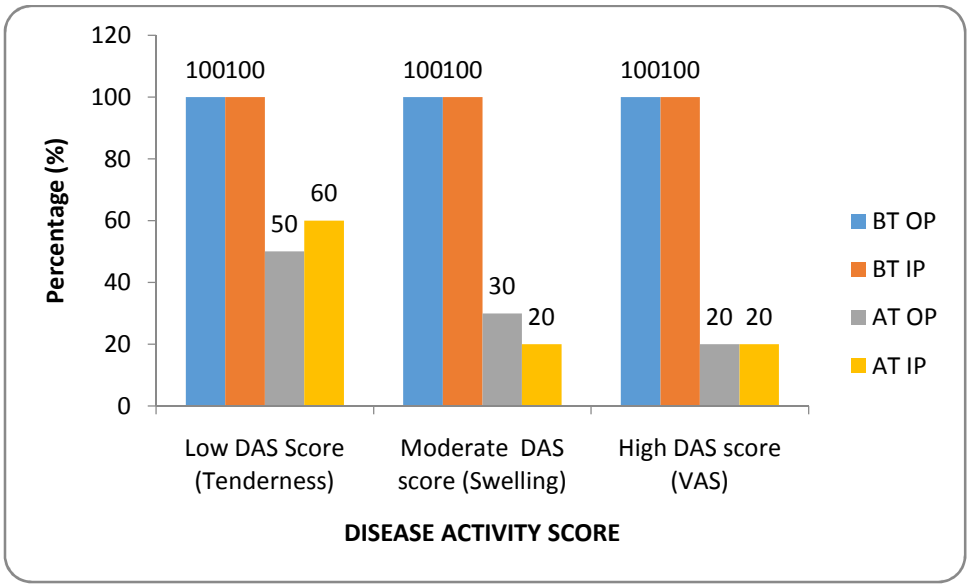
Before Treatment:

Among 20 Out patients, and 20 In patients 100% of cases were with High disease activity.

After Treatment:

Among 20 Out patients, 50% of cases with Low disease activity 30% of cases with Moderate disease activity and 20% of cases with High disease activity. Among 20 In

patients, 60% of cases with Low disease activity, 20% of cases with Moderate disease activity and 20% of cases with High disease activity.



24 ASSESSMENT OF OUTCOME:

Table-24 Illustrates the Assessment of Outcome and its percentage.

TABLE-24

ASSESSMENT OF OUTCOME

Sl. No.	Assessment of Outcome	Before Treatment				After Treatment			
		Out Patients (OP)		In Patients (IP)		Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)	No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Low	-	-	-	-	10	50%	12	60%
2.	Moderate	-	-	-	-	6	30%	4	20%
3.	High	20	100%	20	100%	4	20%	4	20%

Lower disease activity 2.6<DAS28≤3.2.

Moderate disease activity 3.2<DAS28≤5.1.

High disease activity DAS28>5.1.

Reference:

Prevoo ML, Van't Hof MA Kuper HH, Van Leeuwen MA, Van de Putte LB, Van Rie / PL (1995). "Modified disease activity scores that include twenty-eightjoints counts. Development and validation in a prospective longitudinal study of patients with rheumatoid arthritis". Arthritis Rheum. 38 (1):44-8.

Before Treatment:

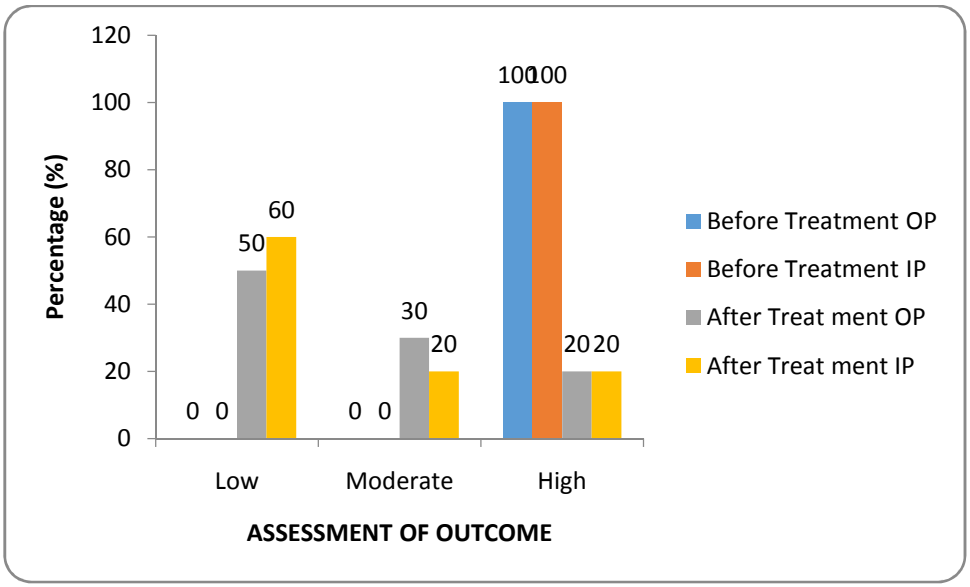
Among 20 Out patients, and 20 In patients 100% of cases were with High disease activity.

After Treatment:

Among 20 Out patients, 50% of cases with Low disease activity 30% of cases with Moderate disease activity and 20% of cases with High disease activity. Among 20 In patients, 60% of cases with Low disease activity, 20% of cases with Moderate disease activity and 20% of cases with High disease activity.

FIGURE-24

ASSESSMENT OF OUTCOME



25 GRADATION OF RESULTS:

Table-25 Illustrates the Gradation of results and its percentage.

TABLE-25

GRADATION OF RESULTS

Sl. No.	Results	Out Patients (OP)		In Patients (IP)	
		No. of Cases	Percentage (%)	No. of Cases	Percentage (%)
1.	Good response	10	50%	12	60%
2.	Moderate response	6	30%	4	20%
3.	Poor response	4	20%	4	20%
	Total	**20**	**100%**	**20**	**100%**

Among 20 Out patients, 50% of cases showed Good response, 30% of cases showed Moderate response and 20% of cases showed Poor response.

Among 20 In patients, 60% of cases showed Good response, 20% of cases showed Moderate response and 20% of cases showed Poor response.

FIGURE-25 GRADATION OF RESULTS

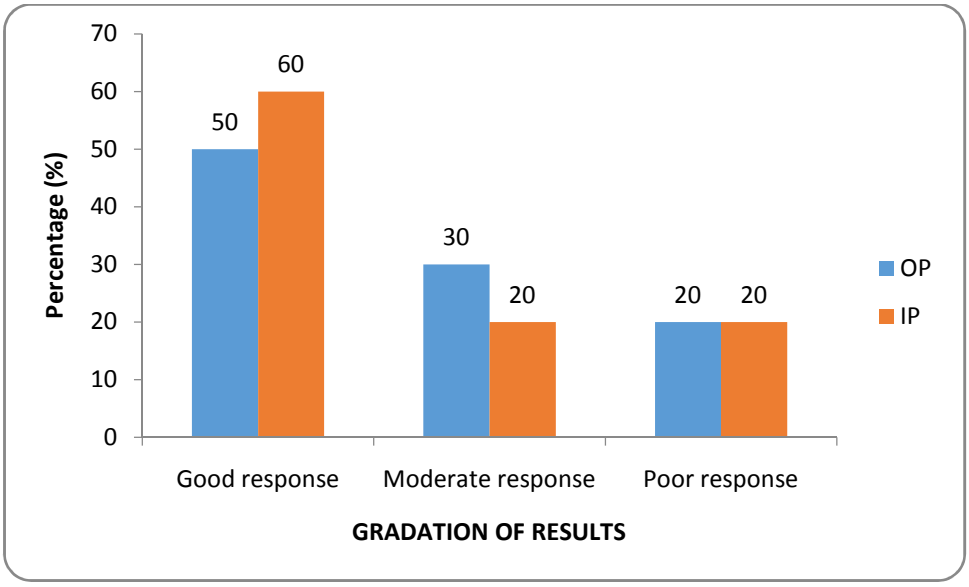


TABLE-26(a)

i) LABORATORY INVESTIGATION OF OUT PATIENTS

Sl. No.	Out Patient No.	HAEMATOLOGICAL REPORT												URINE ANALYSIS					
		BEFORE TREATMENT						AFTER TREATMENT						BEFORE TREATMENT			AFTER TREATMENT		
		TC	DC			ESR (mm)	Hb% (gms)	TC	DC			ESR (mm)	Hb% (gms)	Alb	Sug	Dep- Epi.cells / Pus cells	Alb	Sug	Dep- Epi.cells / Pus cells
			P	L	E	1 Hr.			P	L	E	1 Hr.							
1.	59037	8000	64	38	8	32	13.00	9500	60	37	3	10	13.40	NIL	NIL	1-2 Epi.cells	NIL	NIL	NAD
2.	59417	9200	68	26	2	40	7.00	8600	66	35	4	12	9.00	NIL	NIL	NAD	NIL	NIL	NAD
3.	63452	8000	62	34	4	18	12.60	9000	62	32	6	22	10.40	NIL	NIL	NAD	NIL	NIL	NAD
4.	63573	6700	68	30	2	20	13.20	6400	60	25	2	13	10.20	NIL	NIL	NAD	NIL	NIL	NAD
5.	73028	6000	60	31	9	40	9.80	5000	52	30	8	24	9.40	NIL	NIL	NAD	NIL	NIL	NAD
6.	109410	7800	54	40	6	16	12.50	8000	60	45	5	8	12.80	NIL	NIL	NAD	NIL	NIL	NAD
7.	11640	8000	64	33	3	20	10.20	7800	62	30	2	10	10.20	NIL	NIL	NAD	NIL	NIL	NAD
8.	113090	7800	65	30	5	50	12.80	8000	66	32	4	7	13.00	NIL	NIL	NAD	NIL	NIL	NAD
9.	3237	8200	60	36	8	18	11.00	6200	31	7	9	10	11.20	NIL	NIL	NAD	NIL	NIL	NAD
10.	7590	8100	64	30	5	20	12.60	9800	60	35	5	6	12.90	NIL	NIL	NAD	NIL	NIL	NAD
11.	12172	9400	65	28	7	30	13.00	8700	63	32	5	5	13.20	NIL	NIL	NAD	NIL	NIL	NAD
12.	12554	9100	60	30	2	40	10.00	8900	62	28	3	10	11.00	NIL	NIL	NAD	NIL	NIL	NAD
13.	18807	8400	67	28	5	21	13.50	8100	65	33	2	8	13.70	NIL	NIL	1-2 Epi.cells	NIL	NIL	NAD
14.	19600	8900	64	34	2	30	10.10	9000	62	36	6	24	10.40	NIL	NIL	NAD	NIL	NIL	NAD
15.	22078	8100	60	35	5	20	11.40	8400	64	30	6	10	12.80	NIL	NIL	NAD	NIL	NIL	NAD
16.	22519	8400	67	30	3	30	10.20	8000	59	38	3	30	10.60	NIL	NIL	NAD	NIL	NIL	NAD
17.	243320	9200	60	36	4	50	11.50	8700	60	39	1	6	12.00	NIL	NIL	NAD	NIL	NIL	NAD
18.	22120	8200	61	30	9	40	10.60	8100	54	39	7	10	13.00	NIL	NIL	2-4 Epi.cells	NIL	NIL	NAD
19.	25588	8900	58	34	8	60	13.00	8600	58	36	6	6	13.50	NIL	NIL	NAD	NIL	NIL	NAD
20.	30928	8800	73	25	2	40	11.00	9800	60	36	4	4	12.00	NIL	NIL	NAD	NIL	NIL	NAD

TABLE-26(a)

i). LABORATORY INVESTIGATION (OUT PATIENTS)

Sl. No.	OP No.	Before treatment						After treatment					
		Blood sugar (R)	Blood urea	Serum Cholesterol	Uric acid	Serum Creatinine	Bilirubin	Blood sugar (R)	Blood urea	Serum Cholesterol	Uric acid	Serum Creatinine	Bilirubin
1.	59037	92	20	136	4.10	0.5	0.7	87	20	130	3.80	0.3	0.4
2.	59417	103	19	175	2.20	0.7	0.6	100	21	177	2.80	0.6	0.7
3.	63452	98	19	200	4	0.5	0.4	96	17	200	3.80	0.4	0.4
4.	63573	84	38	190	3.4	0.9	0.5	83	30	180	3.2	0.6	0.4
5.	73028	72	21	120	4.2	0.6	0.7	70	20	118	4	0.5	0.6
6.	109410	93	33	149	4	0.8	0.5	80	15	120	2	0.4	0.3
7.	11640	109	18	189	4.20	0.7	0.6	106	16	186	4	0.6	0.4
8.	113090	81	35	156	3.90	0.6	0.8	70	15	140	3.10	0.2	0.4
9.	3237	103	18	180	3.40	0.7	0.7	100	16	160	3.20	0.4	0.5
10.	7590	116	20	186	3.60	0.5	0.6	100	10	146	2	0.3	0.4
11.	12172	97	23	170	3.3	0.9	0.4	90	20	140	2.5	0.4	0.2
12.	125554	102	19	198	3.90	0.6	0.3	98	18	190	3.60	0.5	0.3
13.	18807	75	20	186	3.60	0.4	0.6	70	15	160	3.20	0.3	0.4
14.	19600	97	25	176	4.50	0.6	0.5	99	22	171	4.20	0.5	0.6
15.	22078	65	26	160	3.60	0.4	0.3	60	20	140	3	0.2	0.2
16.	22519	74	30	186	5.30	0.7	0.5	70	33	179	5	0.5	0.4
17.	24320	70	23	170	3.30	0.5	0.4	60	15	120	2	0.2	0.1
18.	22160	91	20	143	3.0	1,0	0.7	90	18	130	4	0.1	0.6
19.	25588	83	36	162	5.20	0.6	0.5	65	30	140	4.60	0.4	0.3
20.	30928	75	24	170	3.80	0.8	0.3	68	20	159	2.40	0.7	0.2

Table-26 (a)

iii). LABORATORY INVESTIGATIONS (OUT PATIENTS)

Sl. No.	OP No.	BEFORE TREATMENT			AFTER TREATMENT	
		RA Factors (IU / ml)	ASO titre (IU / ml)	C – reactive Protein (mg / dl)	RA Factors (IU / ml)	C – reactive Protein (mg / dl)
1.	59037	62.5	90.4	7.8	82.3	7.2
2.	59417	42.6	20.4	9.3	18.4	8.4
3.	63452	54.6	32.8	6.9	30.6	6.8
4.	63573	63.0	18.3	5.2	17.4	5.0
5.	73028	39.6	40.6	8.9	37	8.5
6.	109410	19.00	80.9	6.7	6.2	6.4
7.	11640	70	19.6	3.0	18.4	2.8
8.	113090	18.4	30.4	4.4	15.4	4.2
9.	3237	57.8	5.1	7.9	4.8	6.4
10.	7590	15.7	6.8	8.0	6.9	6.3
11.	12172	256	3.0	4.0	2.8	3.6
12.	12554	49.6	7.4	6.4	6.9	5.3
13.	18807	13.7	46	4.8	30	3.6
14.	19600	16.2	52.3	6.3	51.3	6.2
15.	22078	20.4	9.3	5.3	6.4	3.2
16.	22519	14.3	101.4	6.5	98.2	4.8
17.	24320	18.4	90	5.2	86	4.00
18.	22160	5.8	535	13.6	526	12.4
19.	25588	19.6	120	7.4	102	6.5
20.	30928	15.3	99	13	60	5.4

Reference range:
Rheumatoid factor : Upto 20 IU/ml Serum for ASO : Upto 200 IU/ml CRP : Upto 6 mg/dl

TABLE-26 (b)

i). LABORATORY INVESTIGATION OF IN PATIENTS

Sl. No.	In Patient No.	HAEMOTOLOGICAL REPORT													URINE ANALYSIS					
		BEFORE TREATMENT						AFTER TREATMENT							BEFORE TREATMENT			AFTER TREATMENT		
		TC	DC			ESR (mm)	Hb% (gms)	TC	DC			ESR (mm)	Hb% (gms)		Alb	Sug	Dep- Epi.cells / Pus cells	Alb	Sug	Dep- Epi.cells / Pus cells
			P	L	E	1 Hr.			P	L	E	1 Hr.								
1.	1676	11500	74	16	0.6	100	9.6	12000	74	20	0.3	6	13.4		Nil	Nil	6-8 puscells	Nil	Nil	NAD
2.	1833	8400	64	34	0.2	30	11.2	9300	60	38	0.2	5			Nil	Nil	NAD	Nil	Nil	NAD
3.	1891	9300	60	38	0.2	50	12.4	8100	62	36	0.4	6			Nil	Nil	NAD	Nil	Nil	NAD
4.	1951	13000	78	17	0.3	92	9.8	9800	64	32	0.6	10			Nil	Nil	NAD	Nil	Nil	NAD
5.	1996	8100	60	34	0.4	20	13.5	8400	56	30	0.5	7			Nil	Nil	2 – 3 puscells	Nil	Nil	NAD
6.	2044	9800	63	28	0.5	30	10.6	8600	52	34	0.4	15			Nil	Nil	NAD	Nil	Nil	NAD
7.	2138	8400	65	24	0.3	44	12.9	8600	58	33	0.2	5			Nil	Nil	NAD	Nil	Nil	NAD
8.	2178	8900	61	36	0.2	50	11.6	9300	60	36	0.3	25			Nil	Nil	NAD	Nil	Nil	NAD
9.	2312	9200	70	22	0.4	20	12.4	8400	63	38	0.6	6			Nil	Nil	NAD	Nil	Nil	NAD
10.	2522	8400	68	24	0.5	30	9.10	8400	65	34	0.2	20			Nil	Nil	NAD	Nil	Nil	NAD
11.	2711	9200	64	30	0.2	40	13.6	7500	62	34	0.3	6			Nil	Nil	NAD	Nil	Nil	NAD
12.	2775	8300	70	26	0.4	20	11.5	8600	63	36	0.5	20			Nil	Nil	3 – 2 puscells	Nil	Nil	NAD
13.	3010	8100	61	34	0.5	50	12.4	7800	66	35	0.4	6			Nil	Nil	NAD	Nil	Nil	NAD
14.	3060	9400	67	28	0.3	40	9.8	8900	58	34	0.2	20			Nil	Nil	NAD	Nil	Nil	NAD
15.	3066	8100	63	22	0.5	30	13.3	8600	62	38	0.3	8			Nil	Nil	NAD	Nil	Nil	NAD
16.	3158	9400	69	28	0.4	50	11.5	9000	56	34	0.5	30			Nil	Nil	NAD	Nil	Nil	NAD
17.	262	8200	70	26	0.6	40	10.4	8500	60	30	0.4	15			Nil	Nil	NAD	Nil	Nil	NAD
18.	427	8100	66	30	0.4	30	12.8	8100	54	33	0.6	5			Nil	Nil	NAD	Nil	Nil	NAD
19.	598	8300	62	34	0.4	40	11.2	8700	62	34	0.3	7			Nil	Nil	NAD	Nil	Nil	NAD
20.	1056	8400	70	24	0.3	20	13.0	9200	56	30	0.2	10			Nil	Nil	NAD	Nil	Nil	NAD

26 (b)

ii). LABORATORY INVESTIGATION (IN PATIENTS)

Sl. No.	OP No.	Before treatment						After treatment					
		Blood sugar (R)	Blood urea	Serum Cholesterol	Uric acid	Serum Creatinine	Bilirubin	Blood sugar (R)	Blood urea	Serum Cholesterol	Uric acid	Serum Creatinine	Bilirubin
1.	1676	98	20	189	4.20	0.70	0.80	94	16	187	4.20	0.60	0.40
2.	1833	90	20	146	3.40	0.60	0.90	98	20	147	3.40	0.70	0.90
3.	1891	95	21	182	5.0	0.50	0.40	90	20	180	5.00	0.50	0.40
4.	1951	97	21	191	4.50	0.80	0.70	96	20	180	4.20	0.60	0.40
5.	1996	104	20	188	3.0	0.70	0.90	97	17	159	3.00	0.70	0.80
6.	2044	90	24	189	2.50	0.80	0.50	91	20	183	2.50	0.60	0.50
7.	2138	89	22	179	4.40	0.50	0.60	80	20	160	4.20	0.40	0.30
8.	2178	102	20	150	3.80	0.90	0.50	98	19	138	3.60	0.80	0.60
9.	2312	99	26	160	3.90	0.80	0.40	90	20	150	3.10	0.60	0.20
10.	2522	101	19	148	3.20	0.60	0.70	100	18	147	3.00	0.50	0.60
11.	2711	90	21	160	3.60	0.40	0.30	82	18	148	3.20	0.30	0.10
12.	2775	112	24	158	6.0	0.50	0.40	110	22	150	4.30	0.40	0.30
13.	3010	90	22	148	3.40	0.60	0.80	78	20	120	3.00	0.30	0.40
14.	3060	95	29	180	4.20	0.80	0.30	93	26	178	4.00	0.70	0.20
15.	3066	129	30	184	3.80	0.40	0.70	110	28	169	3.20	0.30	0.60
16.	3158	101	19	147	3.70	0.80	0.60	100	18	146	3.60	0.40	0.50
17.	262	90	24	189	2.50	0.80	0.70	85	22	180	2.00	0.50	0.40
18.	427	104	36	168	4.0	0.40	0.40	100	38	150	3.80	0.30	0.20
19.	598	99	24	199	3.20	0.60	0.30	90	20	180	3.00	0.50	0.20
20.	1056	89	38	158	4.30	0.50	0.90	80	34	148	4.00	0.30	0.50

26(b)

iii). LABORATORY INVESTIGATIONS (IN PATIENTS)

Sl. No.	IP No.	BEFORE TREATMENT			AFTER TREATMENT	
		RA Factors (IU / ml)	ASO titre (IU / ml)	C – reactive Protein (mg / dl)	RA Factors (IU / ml)	C – reactive Protein (mg / dl)
1.	1676	60	285	1.0	20	1.0
2.	1833	17.2	103	3.3	17.1	3.1
3.	1891	11.4	89	2.3	11.2	2.7
4.	1951	19	320	4	16.4	4.6
5.	1996	17.9	140	4.6	17.9	4.6
6.	2044	19.6	60.4	4.7	18.4	4.4
7.	2138	62.4	89	8.6	62.4	7.3
8.	2178	73.6	110.6	8.3	73.2	8.6
9.	2312	18	93	2.4	17.8	2.0
10.	2522	12.6	46.3	2.6	13.9	2.6
11.	2711	19	90.2	2.9	18	2.3
12.	2775	56.3	50	7.2	54.6	6.7
13.	3010	34.6	109	6.8	30.2	6.3
14.	3060	18.2	65.4	1.9	18	1.6
15.	3066	40.6	78.3	5.6	39	6.8
16.	3158	20.8	79.6	8.7	50.6	5.6
17.	262	11.2	88.6	2.3	11.4	2.0
18.	427	250	50	12.6	236	10.6
19.	598	16.4	79.4	3.2	15.6	2.8
20.	1056	20	80.6	2.8	19.7	2.0

Reference range:

Rheumatoid factor : Upto 20 IU/ml Serum for ASO : Upto 200 IU/ml CRP : Upto 6 mg/dl

27 (a) DISEASE ACTIVITY PAIN SCORE (OUT PATIENTS

Sl. No.	OP No.	Before treatment					Afte		
		TJC 28	SJC 28	ESR mm / Hr.	VAS	DAS 28	TJC 28	SJC 28	
1.	59037	4	12	32	70	5.51	2	4	
2.	59417	12	10	40	50	6.11	7	9	
3.	63452	10	9	18	60	5.48	14	16	
4.	63573	12	8	20	50	5.53	8	7	
5.	73028	20	18	40	80	7.41	12	10	
6.	109410	11	10	16	50	5.39	4	4	
7.	11640	13	9	20	60	5.8	3	7	
8.	113090	18	20	50	80	7.5	4	3	
9.	3237	10	9	18	70	5.62	8	12	
10.	7590	14	18	20	50	6.09	2	4	
11.	12172	13	15	30	60	6.33	5	3	
12.	12554	9	12	40	70	6.23	4	4	
13.	18807	13	8	21	50	5.65	2	4	
14.	19600	8	9	30	80	5.94	12	10	
15.	22078	10	11	20	60	5.65	3	6	
16.	22519	15	14	30	50	6.3	12	10	
17.	24320	13	10	50	40	6.21	6	2	
18.	22160	12	16	40	70	6.63	5	4	
19.	25588	16	10	60	40	6.55	4	4	
20.	30928	14	16	40	60	6.65	5	2	

Interpretation:

Low disease activity 2.6<DAS≤3.2 Moderate disease activity 3.2<DAS28≤5.1 High disease

Reference range ESR 5-15mm / hr.

27 (b) DISEASE ACTIVITY PAIN SCORE (IN PATIENTS)

Sl. No.	IP No.	Before treatment						
		TJC 28	SJC 28	ESR mm / Hr.	VAS	DAS 28	TJC 28	SJC 28
1.	1676	8	12	100	40	5.22	4	3
2.	1833	10	14	30	60	6.05	6	4
3.	1891	9	12	50	40	5.96	4	3
4.	1951	13	10	92	60	5.77	11	9
5.	1996	20	14	20	50	6.35	3	4
6.	2044	12	16	30	70	6.43	9	8
7.	2138	16	10	44	50	6.48	4	6
8.	2178	18	13	50	40	6.69	10	14
9.	2312	9	12	20	60	5.6	6	4
10.	2522	12	18	30	40	6.08	10	12
11.	2711	9	8	40	50	5.76	5	3
12.	2775	14	16	20	30	5.74	10	6
13.	3010	11	10	50	40	6.05	3	3
14.	3060	16	14	40	30	6.29	12	14
15.	3066	8	10	30	50	5.56	3	4
16.	3158	18	12	50	40	6.64	14	10
17.	262	10	14	40	50	6.11	8	8
18.	427	12	10	30	40	5.77	4	3
19.	598	11	14	40	50	5.2	2	6
20.	1056	10	8	20	40	5.22	3	4

Interpretation:

Low disease activity 2.6<DAS$\leq$3.2 Moderate disease activity 3.2<DAS28$\leq$5.1 High disease

Reference range ESR 5-15mm / hr

TABLE-28 (a)
CASE SUMMARY - OUT PATIENTS

Sl. No.	OP No.	Name	Age / Sex	Occupation	Duration of illness	Treatment starting date	End of the treatment date	Total Days	Results
1.	59037	மூக்கம்மாள்	40F	Beedi worker	2 Years	11.07.2017	11.08.2017	30	Good
2.	59417	முகைதீன் மீராள்	34F	Housewife	4 Months	12.07.2017	12.08.2017	30	Fair
3.	63452	பாத்திமா	43F	Housewife	1 Year	25.07.2017	25.08.2017	30	Poor
4.	63573	விஜயா	45F	Coolie	1 Year	25.07.2017	25.08.2017	30	Fair
5.	73028	உச்சிமாகாளி	40F	Coolie	2 Months	25.08.2017	25.09.2017	30	Poor
6.	109410	லலிதா	50F	Housewife	4 Months	11.12.2017	11.01.2018	30	Good
7.	11640	வசந்தி	50F	Agricultural labour	2 Months	18.12.2017	18.01.2018	30	Fair
8.	113090	நாராயணி	37F	Housewife	4 Months	22.12.2017	22.01.2018	30	Good
9.	3237	வேல்செல்வி	30F	Housewife	2 Months	08.01.2018	08.02.2018	30	Fair
10.	7590	அகிலாள்	40F	Housewife	2 Years	22.01.2018	22.02.2018	30	Good
11.	12172	காந்திமதி	42F	Sales women	6 Months	05.02.2018	07.03.2018	30	Good
12.	12554	உமாமகேஸ்வரி	47F	Housewife	5 Years	06.02.2018	08.03.2018	30	Fair
13.	18807	மாரியம்மாள்	44F	Coolie	6 Months	23.02.2018	25.03.2018	30	Good
14.	19600	நாகூர்மீரா	50F	Housewife	4 Years	26.02.2018	28.03.2018	30	Poor
15.	22078	ருக்மணி	50F	Agriculutral labour	2 Months	05.03.2018	05.04.2018	30	Good
16.	22519	சரஸ்வதி	49F	Tailor	1 Year	07.03.2018	07.04.2018	30	Poor
17.	24320	ராணி	40F	Housewife	2 Months	12.03.2018	12.04.2018	30	Good
18.	22160	மீனா	50F	Tailor	3 Months	14.03.2018	14.04.2018	30	Fair
19.	25588	சுந்தரி	33F	Housewife	3 Months	16.03.2018	16.04.2018	30	Good
20.	30928	சாந்திநாகராணி	50F	Housewife	3 Months	03.04.2018	03.05.2018	30	Good

TABLE-28 (b)
CASE SUMMARY - IN PATIENT

Sl. No.	IP No.	Name	Age / Sex	Occupation	Duration of illness	DOA	DOD	Total Days		Total Days	Results
								OP	IP		
1.	1676	இசக்கியம்மாள்	18F	Coolie	1 Year	06.06.2017	03.07.2017	2	28	30	Good
2.	1833	லட்சுமி	50F	Agricultural labour	2 Years	24.06.2017	02.07.2017	21	9	30	Good
3.	1891	சலோமி	50F	Agricultural labour	6 months	29.06.2017	01.08.2017		34	30	Good
4.	1951	சுந்தரி	30F	Housewife	4 months	05.07.2017	01.08.2017	2	28	30	Fair
5.	1996	ஜான்	44M	Loadman	4 years	10.07.2017	11.08.2017		33	30	Good
6.	2044	மகேஸ்வரி	48F	Housewife	8 months	17.07.2017	26.07.2017	20	10	30	Fair
7.	2138	பெரியதாய்	47F	Coolie	5 years	28.07.2017	22.08.2017	4	26	30	Good
8.	2178	எஸ்கலின்	50F	Agricultural labour	1 year	02.08.2017	30.08.2017	1	29	30	Poor
9.	2312	வீரம்மாள்	50F	Housewife	6 months	18.08.2017	21.09.2017		35	30	Good
10.	2522	சுப்புலெட்சுமி	49F	Coolie	1 year	09.09.2017	24.10.2017		46	30	Poor
11.	2711	சித்திரக்கணி	45F	Tailor	3 years	06.10.2017	15.11.2017		41	30	Good
12.	2775	பேச்சியம்மாள்	46F	Housewife	7 months	12.10.2017	23.11.2017		43	30	Fair
13.	3010	காளிராஜா	31M	Driver	3 years	09.11.2017	12.12.2017		34	30	Good
14.	3060	பீமாஜான்	40F	Tailor	5 months	15.11.2017	23.12.2017		39	30	Poor
15.	3066	அய்யம்மாள்	48F	Agricultural labour	2 years	16.11.2017	17.12.2017		32	30	Good
16.	3158	பிரகாஷ்	42M	Business	5 months	30.11.2017	24.12.2017	5	25	30	Poor
17.	262	குணவதி	35F	Flower maker	3 months	01.02.2018	13.03.2018		41	30	Fair
18.	427	வள்ளியம்மாள்	45F	Coolie	1 year	17.02.2018	19.03.2018		31	30	Good
19.	598	சந்தனகுமார்	35M	Mason	2 months	06.03.2018	10.04.2018		36	30	Good
20.	1056	லிங்கம்மாள்	50F	Coolie	2 years	18.04.2018	27.04.2018		40	30	Good

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DEFORMITIES

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CHAPTER - VI
DISCUSSION

Vali Azhal Keel Vayu is described in Sabapathy manuscript it is nearly correlated in modern science with Rheumatoid Arthritis (RA). In this clinical trial study totally 40 patients were selected, 20 were treated as Out patient and 20 were treated as In patients with clinical trial drug **'AKKINI CHOORANAM'.**

The follwing results were discussed and given below,

1. Sex distribution:

Among 20 Out patients 0% were Male and 100% were Female. Among 20 In patients, 20% were Male and 80% were Female.

2. Age distribution:

From the above table it is observed that the highest incidence of Vali Azhal Keel Vayu in Out patients is among the age group of 41 to 50 with 60% and 31 to 40 with 35%, 5% were in the age group of 21 to 30 years. Among 20 In patients 5% were in the age group of 11 to 20 years and 21 to 30 years , 20% were in the age group of 31 to 40 years , 70% with the highest incidence in the age group of 41 to 50 years.

3. Kaalam distribution:

Among 20 Out patients, 90% were affected in Pitha Kaalam and 10% were affected in VathaKaalam. Among 20 In patients, 85% were affected in Pitha Kaalam and 15% were affected in Vatha Kaalam.

4. Constitution of body

Vatha Pitha Thegi registered high incidence of Vali Azhal Keel Vayu with 80% OP and 75% IP. Remaining Pitha Vatha Thegi of 20% OP and 15% IP, Pitha Kapham Thegi of 10% In patients.

5. Gunam

Among 20 Out patients, 60% were Rajo gunam and 40% were Thamasa gunam.

Among 20 In patients, 75% were Rajo gunam and 25% were Thamasa gunam.

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6. Religion

Among 20 Out patients, 85% were Hindus, 15% were Muslims. Among 20 In patients, 80% were Hindus,15% were Christians and 5% were Muslims .

7. Paruvakaalam

Among 20 Out patients, 30% of cases were in Munpani Kaalam and Pinpani Kaalam,25% of cases were in Muthuvenil Kaalam and 15% of cases were in Koothir Kaalam. Among 20 In patients, 30% of cases were in Muthuvenil Kaalam, 20% of cases were in Koothir Kaalam and 15% of cases were in Elavenil Kaalam and Kaar Kaalam,10% of cases were in Munpani and Pinpani Kaalam.

8. Thinai

Among 20 Out patients, 50% were in Marutham and 25% were in Neithal and mullai.

Among 20 In patients, 55% were in Marutham and 20% were in Neithal and Mullai, 5% were in Kurinji.

9. Socio economical status

Among 20 Out patients, 35% were in Low income class, 40% were in Middle income class and 25% were in High income class. Among 20 In patients, 40% were in Low income class, 45% were in Middle income class and 15% were in High income class.

10. Food habits

Among 20 Out patients, 15% were Vegetarian and 85% were non – vegetarian.

Among 20 In patients, 0% were vegetarian and 100% were non – vegetarian.

11. Family history

Among 20 Out patients, 55% have positive Family History and 45% don't have any positive Family History. Among 20 In patients, 50% have positive Family History and 50% don't have any positive Family History.

12. Occupation

Among 20 Out patients, 10% Agricultural labours, 15% Coolie, 55% House Wife, 10% Tailor, 5% Beedi worker and Sales women.

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Among 20 In patients, 20% Agricultural labours, 25% Coolie, 20% House Wife, 5% Loadman, 5% Driver,5% Business,5% Flower Maker,5% Manson, 10% Tailor were observed.

13. Clinical manifestations

Among 20 Out patients, 100% cases have arthritis involving more than 3 joints, severe pain and swelling, symmetrical joint involvement,85% have morning stiffness , 45% have depression, 20% have Anorexia, 15% have Rheumatoid nodules. Among 20 In patients, 100% cases have arthritis involving more than 3 joints, severe pain and swelling, symmetrical joint involvement,50% have morning stiffness, 55%% have depression, 25% have rheumatoid nodules, 35% have anorexia.

14. Duration of illness

Among 20 Out patients, Duration of Illness was 5% below 1 year, 10% in 1 to 2 years, 80% in 3 to 4 years and 5% in above 4 years. Among 20 In patients, Duration of Illness was 5% below 1 year, 15% in 1 to 2 years, 10% in 2 to 3 years and 65% in 3 to 4 years,10% above 4 years.

15. Kanmenthiriyam

Among 20 Out patients, 100% cases were affected in Kai, Kaal, 40% cases were affected in Eruvai. Among 20 In patients, 100% cases were affected in Kai, Kaal, 55% cases were affected in Eruvai.

16. Kosam

Among 20 Out patients, 20% were affected in Annamya Kosam, 100% were affected in Pranamaya Kosam,Manomya Kosam and Vinganamaya Kosam. Among 20 In patients, 35% were affected in Annamya Kosam and 100% were affected in Pranamaya Kosam,Manomya Kosam and Vinganamaya Kosam.

17. Gnanendrium

Among 20 Out patients, 65% of the patients were affected in Mei, 15% of the patients were affected in kan.

Among 20 In patients, 45% of the patients were affected in Mei, 10% of the patients were affected in kan.

18. Condition of Mukkutram

a) Condition of Vatham

Viyaanan, Samaanan, were affected in 100% of both Out patients and In patients. Abaanan was affected in 40% of Out patients, and 55% of the In patients,

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Kirugaran was affected in 20% of Out Patients, and 35% of In patients. Devathathan was affected in 90% of Out patients, and 85% of In patients. Koorman was affected in 15% of Out patients and 10% of In patients,

b) Condition of Pitham

Among 20 Out patients, 20% affected in Anala Pitham, 55% in Ranjaga Pitham and 100% in Sathaga Pitham, 15% in Aalosagapitham. Among 20 In patients, 35% affected in Anala Pitham, 50% in Ranjaga Pitham and 100% in Sathaga Pitham, 10% in Aalosagapitham.

c) Condition of Kabam

Kilethagam was affected in 20% of Out patients and 35% of In patients.

Santhigam was affected in 100% of both Out patients and In patients.

19. Involvement of Udal Kattugal (or) Udal thathukkal:

Among 20 Out patients and In patients Saaram, Enbu were affected in 100% of the cases. senneer was affected in 55% of OP and 50% of IP.

20. Conditions of Envagai thervugal

Among 20 Out patients,15 % were affected in Vizhi, 40% were affected in Malam, 65% was affected in Sparisam, Naadi-80% with Vatha Pitham, 20% with Pitha Vatham . Among 20 In patients, 10% were affected in Vizhi, 55% was affected in Malam, 45% was affected in Sparisam, Naadi-75% with Vatha Pitham ,15% with Pitha Vatham and 10% with Pitha Kapham.

21. Neerkuri

Niram was affected in 20% of Out patients and 35% of In patients. Nurai was affected in 10% of Out patients and 15% of In patients.

22. Neikuri

Among 20 Out patients, 80% had Vatha Neer, 20% had Pitha neer.

Among 20 In patients, 75% had Vatha Neer, 25% had Pitha Neer.

23. Disease Activity Score

Before Treatment:

Among 20 Out patients, and 20 In patients 100% of cases were with High disease activity.

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After Treatment:

Among 20 Out patients, 50% of cases with Low disease activity 30% of cases with Moderate disease activity and 20% of cases with High disease activity. Among 20 In patients, 60% of cases with Low disease activity, 20% of cases with Moderate disease activity and 20% of cases with High disease activity.

24. Assessment of Outcome:

Before Treatment:

Among 20 Out patients, and 20 In patients 100% of cases were with High disease activity.

After Treatment:

Among 20 Out patients, 50% of cases with Low disease activity 30% of cases with Moderate disease activity and 20% of cases with High disease activity. Among 20 In patients, 60% of cases with Low disease activity, 20% of cases with Moderate disease activity and 20% of cases with High disease activity.

Result:

Stastitical analysis has shown DAS 28 score among OP & IP was found to be highly significant at $P<0.0001$ from Table:

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Statistical analysed using student's paired t-test using the Prism Graph pad software. The results were expressed as Mean ± standard deviation and p values <0.001 was considered as statistically significant.

DAS 28 comparison through statistical analysis

Sl.No	Parameters	Mean ± SD		T value	P value	Result
		BT	AT			
1.	Outpatient	6.13±0.60	3.91±1.01	8.401	<0.0001	HS
2.	Inpatient	5.99±0.42	3.92±1.04	8.26	<0.0001	HS

(n=20) (Listwise)

BT – Before Treatment

AT – After Treatment

HS – Highly significant

25. Gradation of Results:

Among 20 Out patients, 50% of cases showed Good response, 30% of cases showed Moderate response and 20% of cases showed Poor response.

Among 20 In patients, 60% of cases showed Good response, 20% of cases showed Moderate response and 20% of cases showed Poor response.

All the 40 patients were treated with the clinical trial medicine **AKKINI CHOORANAM** 4.2gms BID with sugar for 30 days. Thus at the end of the result, the clinical trial drug showed good clinical improvement of the disease

CHAPTER – VII

SUMMARY

An open labeled randomized clinical study on **'VALI AZHAL KEEL VAYU'** with reference to its Aetiology, Pathogenesis, Clinical features, diagnosis, investigations and treatment were conducted at Department of Pothu Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai.

This clinical study of **Vali Azhal Keel Vayu** is done on the basis of reference in Sabapathi manuscript in **Noinadal, Noi mudhal naadal Thiratu Part-II, Pg. No. 623.** The disease is correlated with Rhematioid arthritis.

The trial drug chosen for the clinical study – **AKKINI CHOORANAM** Dosage of 4.2 gms twice a day with sugar after food (Ref. Koshayi Anuboga Vaithiya Bramma Ragasiyam Part – II, Pg. No. 104).

The aetiology, pathology, pathogenesis, clinical features, classification and prognosis of the disease were collected from a number of literatures both in Siddha system as well as in modern system of medicine.

For this study, Out of 40 patients, 20 patients were diagnosed clinically and admitted in the In patients ward and treated with trial medicines. Another twenty patients was treated Out patients department.

The selection of patients and management of patients during admission and after treatment is carried out under the supervision of Professor, Associate Professor, Lecturers of Department of Pothu Maruthuvam.

A case sheet proforma is prepared with particulars focus on Siddha and Modern clinical parameters.

Separate cases sheets were maintained for each patient in In-patient ward and their vital signs and symptoms were monitored and recorded daily. The patients were treated with the trial drug **AKKINI CHOORANAM.**

Routine blood examinations, Urine analysis, other specific investigations and radiological investigations were done by modern scientific methods and were considered for diagnosis and to follow the progress of the patients.

Siddha diagnosis is made up with the help of Ezhu Udal Kattugal and Envagai Thervugal.

Since Vali Azhal Keel Vayu is a chronic disease, it requires treatment for minimum thirty days to minimize the severe pain, tenderness and swelling with slight disappearance of stiffness. The patient is advised to follow up the treatment in Out patient Department.

From this study the following datas were clear that disease was more common in female than male. Maximum incidence was in Pitha Kaalam. Clinically marked reduction in the symptoms along with increase sense of well being, decrease in ESR, decrease in symptoms, decrease in the DISEASE ACTIVITY PAIN SCORE OF 28 JOINTS was noted.

The patients were observed for a period of 3 months during and after the course of treatment. No signs of complications were observed. Clinically no toxic effects were noticed during the treatment period. The pharamacological evaluation of **AKKINI CHOORANAM** showed significant anti inflammatory and analgesic activity. No acute toxicity, side effects were noted.

Bio chemical analysis of **AKKINI CHOORANAM** showed the presence of calcium, sulphate, chloride, ferrous iron, starch, tannic acid, amino acids and unsaturated compound

CHAPTER-VIII

CONCLUSION

In Sabapathy Manuscript was described under **Vali Azhal Keel Vayu** explains the clinical conditions as rheumatoid arthritis. The lines of this version are well analysed under Siddha and modern parameters and the cases are thoroughly examined with clinical and bio chemical report.

Treatment is given on the basis of Mukkutra Theory. The deranged kutrams are made to normal by the trial medicine.

The trial medicine, **AKKINI CHOORANAM** has the taste of kaarupu, inippu according to the taste of individual ingredients.

Kaarpu suvai - Has its function of relieving indigestion, flatulence and constipation.

Inippu suvai - Regulates the vitiation of Vatham and Pitham.

Thus the trial drug based of its suvai acts as an effective anti-arthritic drug. Almost all the cases treated with the trial drug shows considerable improvements. Further follow up of all patients showed a significant relief of their symptoms.

- 50 % of Out patients and 60% of In patients showed Good response.
- 30% of Out patients and 20% of In patients showed Moderate response.
- 20% of Out patients and 20% of In patients showed Poor response.

I conclude that this trial drug **'AKKINI CHOORANAM'** has improved the clinical symptoms of the patients and assured them a better standard of living.

INGREDIENTS OF AKKINI CHOORANAM

Seeragam

Thippili

Chukku

Lavangam

AKKINI CHOORANAM

ANNEXURE-I

PREPARATION AND PROPERTIES OF TRIAL MEDICINE

அக்கினிச்சூரணம்

- (Reference: Koshayi Anuboga Vaithiya Bramma Ragasiyam, Part-II Page No.104)

சுக்கு, சீரகம், திப்பிலி, லவங்கம் இவைகள் வகைக்குப்பலம் - 1 இளவறுப்பாய் வறுத்து சூரணித்து வராகனிடை வீதம் தினம்-2 தடவை நெய்யிலாவது சீனியிலாவது கொடுத்துவர அக்கினி மந்தம், வயிற்றுவலி, குன்மம், வாதம் முதலிய கொடிய ரோகங்கள் குணமாகும்.

S.No	Drugs	Botanical Name	Action	Dose	Part used	Phytochemicals
01	**SEERAGAM**	*Cuminum cyminum Linn Apiaceae*	Anti inflammatory, carminative, stomachic, Astringent cooling	35g	Fruits	Apigenin-7-glucoside Luteolin-7-0-glucoside Essential oil
02	**THIPPILI**	*Piper longum Linn Piperaceae*	Stimulant Carminative Vermifuge	35g	Flower bud	Resin, Volatile oil, starch, Fattyoil, Piperine
03	**CHUKKU**	*Zingiber officinalis Rose Zingiberaceae*	Stimulant Stomachic Carminative	35g	Rhizome	Volatile oil Camphene Phellandrene Zingiberingeineol Borneol, oleo resin
04	**LAVANGAM**	*Szygium aromaticum.Linn Myrtaceae*	Antispasmodic Carminative Stomachic	35g	Flower bud	Alkaloids Sapanin Flavonoids Terpenoids Tanin

Dose : 4.2 gms (1 varaganedai) twice a day

Adjuvant : Sugar, Ghee

Duration : 30 days

Seeragam

சுவை : கார்ப்பு, இனிப்பு

தன்மை: தட்பம்

பிரிவு : இனிப்பு

பொதுக்குணம்

பித்தமெனு மந்திரியைப் பின்னப் படுத்தியவன்

சத்துருவை யுந்துறந்து சாதித்து – மத்தனெனும்

ராசனையு மீவென்று நண்பைப் பலப்படுத்தி

போசனகு டாரிசெயும் போர்.

இதனால் அழல்போம், வயிற்றுவலி, வாய்நோய், ஈரல்நோய், காசம், கல்லடைப்பு, குருதிக்கழிச்சல், இரைப்பு, கம்மல், மூக்குநீர்பாய்தல், வெறி, வளிநோய்கள் இவை விலகும்.

Thippili

சுவை : இனிப்பு

தன்மை: தட்பம்

பிரிவு : இனிப்பு

பொதுக்குணம்

கட்டி யெதிர்நின்று கடுநோயெல் லாம்பணியும்

திட்டி வினையகலும் தேகமெத்த – புட்டியாம்

மாமனுக்கு மாமனென மற்றவர்க்கு மற்றவனாங்

காமமெனுந் திப்பிலிக்கும் கை.

கடுமையான ஐயப்பிணிகளை அகற்றி, உடற்கு வன்மையை அளித்திடும்.

Chukku

சுவை : கார்ப்பு

தன்மை: வெப்பம்

பிரிவு : கார்ப்பு

பொதுக்குணம்

சூலைமந்தம் நெஞ்செரிப்பு தோடமேப் பம்மழலை

மூலம் இரைப்பிருமல் மூக்குநீர் – வாலகப

தோடமதி சாரந் தொடர்வாத குன்மநீர்த்

தோடம்ஆ மம்போக்குஞ் சுக்கு.

சுக்கினால் செரியாமை, மார்பெரிச்சல், புளியேப்பம், வெப்பம், கீழ்வாய்நோய், இரைப்பு, இருமல், கழிச்சல், நீரேற்றம், குன்மம், வயிற்றுப்பிசம், காதுக்குத்தல், முகநோய், தலைநோய், குலைவலி, பாண்டு, வயிற்றுக்குத்தல், ஐயசுரம் போம்.

Lavangam

சுவை : கார்ப்பு

தன்மை: வெப்பம்

பிரிவு : கார்ப்பு

பொதுக்குணம்

பித்தமயக்கம் பேதியொடு வாந்தியும்போம்

சுத்திவிரத் தக்கடுப்புந் தோன்றுமோ – மெத்த

இலவங்கங் கொண்டவருக் கேற் சுகமாகும்

மலமங்கே கட்டுமென வாழ்த்து.

இதனால் மயக்கம், பேதி, வாந்தி, குருதிக்கழிச்சல், நாட்பட்ட கழிச்சல், எருவாய்க்கடுப்பு, செவிநோய், சிவந்தமச்சம், கறுத்த மச்சம், கண்ணால் பூ, படைகள் ஆகியவற்றை நீக்கும்.

PURIFICATIONS OF RAW DRUGS

- Seeragam

  Soak in Ca(OH)2 water on 21 hours then sun dried

- Thippili

  Soak in lime juice and make it dry

- Chukku

  Peel off the outer layer and cut small pieces allow it to dried on the shade light.

- Lavangam

  Just remove the adulterant and make it dried on the shade light.

ANNEXURE-II

BIO – CHEMICAL ANALYSIS OF AKKINI CHOORANAM

PREPARATION OF THE EXTRACT

5gms of the drug was weighed accurately and placed in 250ml clean beaker. Then 50ml of distilled water was added and dissolved well. Then it was boiled well for about 10 minutes. It was cooled and filtered in a 100ml volumetric flask and then it was makeup to 100ml with distilled water. This fluid was taken for analysis.

QUALITATIVE ANALYSIS

S.NO	EXPERIMENT	OBSERVATION	INFERENCE
1	**TEST FOR CALCIUM** 2ml of the above prepared extract is taken in a clean test tube. To this add 2ml of 4% Ammonium oxalate solution.	A white precipitate is formed.	Indicates the presence of calcium.
2	**TEST FOR SULPHATE:** 2ml of the extract is added to 5% barium chloride solution.	A white precipitate is formed.	Indicates the presence of sulphate.
3	**TEST FOR CHLORIDE:** The extract is added with silver nitrate solution	A white precipitate is formed.	Indicates the presence of chloride.
4	**TEST FOR CARBONATE:** The substance is treated with concentrated Hcl	No brisk effervescence is formed.	Absence of carbonate.
5	**TEST FOR STARCH:** The extract is added with weak iodine solution.	Blue colour is formed.	Indicate the presence of starch.
6	**TEST FOR IRON FERRIC:** The extract is acidified with Glacial acetic acid and potassium ferro cyanide.	No blue colour is formed.	Absence of ferric iron.

7	**TEST FOR IRON FERROUS:** The extract is treated with concentrated Nitric acid and Ammonium thio cyanate solution.	Blood red colour is formed.	Indicates the presence of ferrous iron
8	**TEST FOR PHOSPHATE:** The extract is treated with Ammonium Molybdate and concentrated nitric acid.	No yellow precipitate is formed.	Absence of phosphate.
9	**TEST FOR ALBUMIN:** The extract is treated with Esbach's Reagent.	No yellow precipitate is formed.	Absence of albumin.
10	**TEST FOR TANNIC ACID:** The extract is treated with ferric chloride.	Blue black precipitate is formed.	Indicates the presence of tannic acid.
11	**TEST FOR UNSATURATION:** Potassium permanganate solution is added to the extract.	It gets decolorized.	Indicates the presence of unsaturated compound.
12	**TEST FOR THE REDUCING SUGAR:** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.	No colour change occurs.	Absence of reducing sugar.
13	**TEST FOR AMINO ACID:** One or two drops of the extract is placed on a filter paper and dried it well. After drying, 1% Ninhydrin is sprayed over the same and dried it well.	Violet colour is formed.	Indicate the presence of amino acid.
14	**TEST FOR ZINC:** The extract is treated with potassium Ferrocyanide.	No white precipitate is formed.	Absence of zinc.

INFERENCE:

Indicates the presence of *Calcium, sulfate, Chloride, Starch, Ferrous iron, Tannic acid, Unsaturated compound and Amino acid.*

ANTI-INFLAMMATORY ACTIVITY OF SIDDHA FORMULATION AKKINI CHOORANAM

The anti-inflammatory activities of **siddha formulation AKKINI CHOORANAM** at a dose of 100 and 200mg/kg were evaluated using carrageenan-induced paw edema method. The inflammation was readily produced in the form of edema with the help of irritant such as carrageenan. Carrageenan is a sulphated polysaccharide obtained from sea weed (Rhodophyceae) and when injected cause the release of prostaglandins by the way it produces inflammation and edema.

REQUIREMENTS:

Animal : Albino rat (180-200 g)

Drugs and chemicals : Carrageenan (1%w/v), Diclofenac sodium (standard),

Carboxy methyl cellulose (1%w/v),

Digital plethysmo meter. U G O Basile (Italy)

Test compounds : siddha formulation **AKKINI CHOORANAM**

METHOD:

Anti-inflammatory activity was performed by the following procedure of Bhandri et al(1) The animals were divided into 4 groups each having six animals. A freshly prepared suspension of carrageenan (1% w/v , 0.1 ml) was injected to the planter region of left hind paw of each rat. One group was kept as control and the animals of the other groups were pretreated with the siddha formulation **AKKINI CHOORANAM** test Compounds dissolved with 2 ml sterile water given through orally 30 min before the carrageenan treatment. The paw volumes of the test compounds, standard and control groups were measured at 60,240,360 minutes of carrageenan treatment with the help of Digital plethysmometer (Ugo basile, Italy). Mean increase in paw volume was measured and the percentage of inhibition was calculated.

% Anti-inflammatory activity = (Vc-Vt / Vc)* x *100

Where, ***Vt***-mean increase in paw volume in rats treated with test compounds,

Vc-mean increase in paw volume in control group of rats.

TABLE No.1

ANTI-INFLAMMATORY ACTIVITY OF SIDDHA FORMULATION AKKINI CHOORANAM

Treatment	Dose (mg/kg)	Paw volume(ml) as measured by mercury displacement at 6 hour	Percentage inhibition of paw edema
Group I Normal saline	10ml/kg orally	5.63±0.98	-
Group II Std	10mg/kg I.P.Diclofenac sodium	1.71±0.48	69.62%*a
Group III AKKINI CHOORANAM	100mg/kg.Orally.	2.10±0.48	62.69%*a
Group IV AKKINI CHOORANAM	200mg/kg.Orally.	1.95±0.51	65.36%*a

* Data are expressed as Mean ± S.E.M.

*Data were analyzed by one way ANOVA followed by Newman's keul's multiple range tests, to determine the significance of the difference between the control group and rats treated with the test compounds.

*a Values were significantly different from normal control at P< 0.01.

Results

Anti- inflammatory activity

Both doses of siddha formulation **AKKINI CHOORANAM** at 100mg/kg and 200mg/kg were tested for their Anti- inflammatory activity by using carrageenan Induced rat paw edema method and the results are tabulated in table no 1. The results reveals that both doses of siddha formulation **AKKINI CHOORANAM** at 100mg/kg and 200mg/kg doses possesses significant Anti- inflammatory activity when compared to control group at p<0.01.

ANALGESIC ACTIVITY

Analgesic activity of siddha formulation **AKKINI CHOORANAM** was evaluated by acetic acid induced writhing reflux in mice. Painful reaction in animals may be produced by the chemicals such as phenylquinone, bradykinin etc. Like that, acetic acid pain reaction which is characterized as a writhing response. Construction of abdomen, turning of trunk (twist) and extension of hind legs are taken as reaction to chemically induced pain. Analgesics (both narcotic and non-narcotic) inhibit writhing response.

REQUIREMENTS:

Animal : Swiss albino mice (20-25g) either sex

Drugs and chemicals : Diclofenac sodium (standard),
Acetic acid (1%v/v), **AKKINI CHOORANAM**

METHOD:

TREATMENT PROTOCOL

Group-1 Treated as normal control received 10ml/kg of normal saline through orally.

Group-2 Treated as Standard control received 10mg/kg of diclofenac sodium through
Intraperitonealy.

Group-3 Treated as treatment control received 100mg/kg of **AKKINI CHOORANAM** administered through orally.

Group-4 Treated as treatment control received 200mg/kg of **AKKINI CHOORANAM** administered through orally.

Siddha formulation **AKKINI CHOORANAM** was administered one hour prior to the acetic acid administration. Note the onset on writhing. Record the numbers of abdominal contractions, trunk twist and extension of hind limbs as well as the number of animals showing such response during a period of 10 minutes were noted.

STATISTICS:

Data are expressed as mean ± SEM; data analyzed by one way ANOVA followed by Newman's keul's multiple range tests to determine the significance of the difference between the control group and rats treated with the extracts.

* Values were considered significant at P< 0.01.

TABLE No.1

ANALGESIC ACTIVITY OF AKKINI CHOORANAM BY ACETIC ACID INDUCED WRITHING REFLUX IN MICE

Treatment	Dose (mg/kg)	No. of writhing	% reduction in reaction time
Group I Normal saline	Inject 1%v/v acetic acid 1ml/100g of body weight	42.3±2.86	-
Group II Std	10mg/kg I.P.Diclofenac sodium	11.4±0.82	73.04%**
Group III AKKINI CHOORANAM	100mg/kg Administered through orally.	15.3±1.54	63.82%**
Group IV AKKINI CHOORANAM	200mg/kg Administered through orally	13.7±1.25	67.61%**

Values are expressed as mean±SEM

Values were find out by using one-way ANOVA followed by Newman's keuls multiple range tests.

** Values were considered significant at P< 0.01.

RESULTS

The table values show that analgesic activity of **AKKINI CHOORANAM** by acetic acid induced writhing reflex. The results reveals that both dose of **AKKINI CHOORANAM** possess significant analgesic activity at p<0.01.

ACUTE TOXICITY STUDY OF AKKINI CHOORANAM

Determination of acute oral toxicity is usually the initial screening step in the assessment and evaluation of the toxic characteristics of all compounds. The types of toxicity tests which are routinely performed by pharmaceutical manufacturers in the investigation of a new drug involve acute, sub-acute and chronic toxicity. Acute toxicity is involved in estimation of LD_{50} (the dose which has proved to be lethal (causing death) to 50% of the tested group of animals)

(Shetty Akhila, *et al.,* 2007).(1)

Method: Acute oral toxicity of **AKKINI CHOORANAM** is carried out as per the guidelines Organization of Economic Co-operation and Development (OECD) -423 guidelines after the animal ethical clearance from Institutional Animal Ethics Committee.

The albino mice are fasted over night and provided only water, after which the **AKKINI CHOORANAM** is administered by gastric intubations to the relevant group of animals orally at the dose of 50 $mg.kg^{-1}$ body weight in Tween-80. The animals are then observed for 14 days and maintained with normal food. A mortality rate of 2 or 3 animals in 14 days is recorded and the dose is said to be toxic dose. But when mortality of one animal is observed, then the same dose is repeated again for confirmation. However, if mortality is not observed, the procedure is repeated for further higher doses such as 300 and 2,000 $mg.kg^{-1}$ body weight. Toxic symptoms are observed for 72 hrs including behavioral changes, locomotion, convulsions and mortality (Shah Ayub, 1997, Bürger, 2005).(2,3).

Cage Side Observations

Observations include changes in skin and fur, eyes and mucous membranes, and also respiratory, circulatory, autonomic and central nervous systems, and somatomotor activity and behavior pattern. Special attention is directed for the observation of tremors, convulsions, salivation, diarrhea, lethargy, sleep and coma.

Body Weight, Food and Water Intake

Body weight, food and water intake are recorded at two-day intervals.

Pathology

Surviving animals are fasted overnight, weighed and humanely killed on the 15th day using anesthetic ether. All test animals are subjected to gross necropsy.

Subchronic test for AKKINI CHOORANAM

This experiment evaluates the toxicity potential of**AKKINI CHOORANAM**.

Method: Male and female Wistar rats weighing 180 ± 10 g are used for the present study. The animals are divided into five groups of six animals each. The dose of the preparation is calculated based on the body weight of the animal. The animals in Group I are administered with a single daily dose of 0.5 ml of Tween 80 orally for 20 days. The animals in Group II are administered with 50 $mg.kg^{-1}$b.w. of the **AKKINI CHOORANAM**orally once daily for 20 days. The animals in Group III are administered with 100 $mg.kg^{-1}$b.w. of the **AKKINI CHOORANAM** orally once daily for 20 days. The animals in Group IV and V are administered once daily with 200 and 400 $mg.kg^{-1}$b.w. of the **AKKINI CHOORANAM** respectively for 20 days orally (Pieme,*et al* 2006, Joshi, *et al* 2007, Mythilypriya, *et al.,* 2007).(4,5,6)The animals are then weighed every five days, from the start of the treatment, to record the weight variation. At the end of the treatment, blood samples are collected by puncturing retro orbital plexus after mild anesthesia for biochemical analysis. The collected blood sample is centrifuged within 5 min of collection at 4000 g for 10 min to obtain plasma, which is analyzed for total cholesterol, total triglyceride, HDL-cholesterol levels,LDL-cholesterol,plasma glucose, alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatinine and urea.

Results

Acute toxicity study with AKKINI CHOORANAM

The acute toxicity of **AKKINI CHOORANAM**was evaluated usingOECD-423 guidelines. There was no mortality or morbidity observed in animals through the 15-days period following single oral administration at all selected dose levels of the **AKKINI CHOORANAM**(Table-1). The animals did not show any changes in the general appearance during the observation period. Morphological characteristics such as fur, skin, eyes and nose appeared normal. No tremors, convulsion, salivation, diarrhea, lethargy or unusual behaviors such as self mutilation, walking backward and

so forth were observed. Gait and posture, reactivity to handling or sensory stimuli, grip strength was also normal.

	Dose ($mg.kg^{-1}$)	Sign of Toxicity ($ST.NB^{-1}$)	Mortality ($D.S^{-1}$)
Group I	0	0/3	0/3
Group II	300	0/3	0/3
Group III	2000	0/3	3/3

Table.1. Acute toxicity studyof **AKKINI CHOORANAM** on experimental mice. The acute toxicityof **AKKINI CHOORANAM** on experimental mice was tested using OECD-423 guidelines, where ST- sign of toxicity; NB- normal behaviour; D-died; S- survive. Values are expressed as number of animals (n=3).

Effect of AKKINI CHOORANAM inSubchronic Toxicity

AKKINI CHOORANAMwere evaluated for subchronic toxicity.

Effect of AKKINI CHOORANAM on body weight changes in rats

The effect of **AKKINI CHOORANAM**was observed for their effect on the body weight changes from the study it was observed that, there was significant increase ($p<0.05$) in body weight in all the animals observed. The results are shown in Table.2.

Treatment	Day 1	Day 5	Day 10	Day 20
Control	185.14±5.4	190.45 ±6.14	199.10 ±6.30	199.6±6.28
AKKINI CHOORANAM 50 $mg.kg^{-1}$	196.34 ±6.2	199.35 ±6.45	200.48 ±6.75	200.30±6.82*
AKKINI CHOORANAM 100 $mg.kg^{-1}$	189.36 ±6.0	196.48 ±6.40	198.30 ±6.54	200.89±6.68*
AKKINI CHOORANAM 200 $mg.kg^{-1}$	198.25 ±7.0	200.25±6.32	200.48 ±6.58**	208.35±6.70**
AKKINI CHOORANAM	189.54 ±6.34	196.40 ±6.60	198.15 ±6.65**	206.52±6.72**

400 mg.kg^{-1}				

Table.2.The effects of **AKKINI CHOORANAM** on body weight changes in rats. A study on the effects of **AKKINI CHOORANAM** on body weight changes in rats was carried out.. where, group I animals (GPI) were treated with normal saline (5 ml.kg^{-1}), group II animals (GPII) with 50 mg.kg^{-1} of **AKKINI CHOORANAM**, group III animals (GPIII) with 100 mg.kg^{-1} of **AKKINI CHOORANAM**, group IV animals (GPIV) with 200 mg.kg^{-1} of **AKKINI CHOORANAM**, group V animals (GPV) with 400 mg.kg^{-1}**AKKINI CHOORANAM**. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01 *P<0.05.

Effect of AKKINI CHOORANAM on kidney,heart, liver and brain in rats. The effects of **AKKINI CHOORANAM** on kidney, heart, liver and brainof the rats were observed.From the study it was clear that, significant (p<0.01) changes in the weights of various organs of the animals occurred with higher doses of the extract (400 mg.kg^{-1}bwt), but macroscopic examinations did not show any changes in colour of the organs of the treated animals compared with the control. The results are shown in Table.3.

Treatment	**Heart (gms)**	**Kidney (gms)**	**Liver (gms)**	**Brain (gms)**
Control	0.35 ± 0.04	0.72± 0.03	3.33± 0.14	0.74± 0.05
AKKINI CHOORANAM @50 mg.kg^{-1}	0.36± 0.05	0.82± 0.05	3.43± 0.19	0.72± 0.03
AKKINI CHOORANAM @100 mg.kg^{-1}	0.39± 0.06	0.82± 0.04	3.45±0.21	0.70± 0.08
AKKINI CHOORANAM @ 200 mg.kg^{-1}	0.34± 0.03	0.75± 0.02	3.37± 0.22	0.78± 0.09

AKKINI CHOORANAM @400 mg.kg^{-1}	0.37± 0.05	0.74± 0.02	3.38± 0.15	0.77± 0.12

Table.3.The effects of **AKKINI CHOORANAM**on kidney, heart, liver and brainof the rats. A study on the effects of **AKKINI CHOORANAM**on kidney, heart, liver and brainof the rats was tested. where, group I animals (GPI) treated with normal saline (5 ml.kg^{-1}), group II animals (GPII) with 50 mg.kg^{-1} of **AKKINI CHOORANAM**, group III animals (GPIII) with 100 mg.kg^{-1} of **AKKINI CHOORANAM**, group IV animals (GPIV) with 200 mg.kg^{-1} of **AKKINI CHOORANAM**, group V animals (GPV) with 400 mg.kg^{-1}**AKKINI CHOORANAM**. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01.

Effect of AKKINI CHOORANAM on biochemical profiles of rats

The effect of **AKKINI CHOORANAM** on various biochemical parameters of the experimental animal 'rats' were tested.From the study it was evident that, there was significant decrease ($p<0.05$) in the plasma glucose level in treated rats especially at higher dose (400 mg.kg^{-1}) compared with control rats. The control rats were administered only with 5 ml of normal saline. Significant decrease ($p<0.05$) in the plasma total cholesterol (TC), triglyceride (TG) and LDL-cholesterol levels were observed. But a significant increase ($p<0.05$) in HDL-cholesterol levels were observed in all the treated animals compared with the control animals. AST, ALT and ALP levels were also normal in the **AKKINI CHOORANAM** treated animals. From the results of biochemical study there was no evidence of severe toxicity associated with the administration of higher concentration of **AKKINI CHOORANAM**. The results are shown in Table.4.

Treatment	**Glucose (mg.dl^{-1})**	**Cholesterol (mg.dl^{-1})**	**Triglyceride (mg.dl^{-1})**	**HDL (mg.dl^{-1})**	**LDL (mg.dl^{-1})**
Control	89.42±1.72	34.05± 0.62	29.25±1.43	143.45±3.15	81.30±1.85
AKKINI CHOORANAM @ 50	87.50±1.60	20.30± 0.33*	12.36± 0.85*	181.40±3.65*	67.75±1.38

mg.kg^{-1}					
AKKINI CHOORANAM@ 100 mg.kg^{-1}	85.44±1.50	18.65± 0.33*	14.32± 0.90*	170.30±3.40*	64.54±1.30
AKKINI CHOORANAM @ 200 mg.kg^{-1}	84.30±1.33**	24.20± 0.35	15.40± 0.92*	189.34±3.70*	41.52±1.18
AKKINI CHOORANAM @ 400 mg.kg^{-1}	87.28±1.41**	25.45± 0.45	17.30±1.15*	187.24±3.66*	40.30±1.05

Table.4.The effect of **AKKINI CHOORANAM** on biochemical parameters such as glucose, cholesterol, triglyceride, HDL and LDL. A study on the effect of **AKKINI CHOORANAM**on biochemical parameters such as glucose, cholesterol, triglyceride, HDL and LDL in rats was tested.where, group I animals (GPI) treated with normal saline (5 ml.kg^{-1}), group II animals (GPII) with 50 mg.kg^{-1} of **AKKINI CHOORANAM**, group III animals (GPIII) with 100 mg.kg^{-1} of **AKKINI CHOORANAM**, group IV animals (GPIV) with 200 mg.kg^{-1} of, group V animals (GPV) with 400 mg.kg^{-1}**AKKINI CHOORANAM**. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01 *P<0.05

Treatment	**AST (IU.l^{-1})**	**ALT (IU.l^{-1})**	**ALP (IU.l^{-1})**	**TP (g.l^{-1})**	**ALBUMIN (g.l^{-1})**
Control	320.3±11.58	75.4± 3.44	255.35± 8.62	63.36± 3.26	33.30±2.47
AKKINI CHOORANAM @50 mg.kg^{-1}	310.4±10.50**	73.3± 2.92**	267.15±8.73**	63.30±3.18	30.24±2.32
AKKINI CHOORANAM @ 100 mg.kg^{-1}	309.5±10.62**	70.3±2.94**	268.38±8.35**	73.12±3.78	31.30±2.43
AKKINI CHOORANAM @ 200 mg.kg^{-1}	308.5±9.92	67.3± 2.40	268.20±8.41	64.35± 3.67	32.28±2.44
AKKINI CHOORANAM @ 400 mg.kg^{-1}	310.4±9.96	67.6±2.48	268.42±8.49	65.30± 3.74	32.64±2.52

Table.5.The effects of **AKKINI CHOORANAM** on biochemical parameters such as AST, ALT, ALP, TP and Albumin in rats. A study on the effects of **AKKINI CHOORANAM** on biochemical parameters such as AST, ALT, ALP, TP and Albumin rats was tested. where, group I animals (GPI) were treated with normal saline (5ml.kg^{-1}), group II animals (GPII) with 50 mg.kg^{-1} of HAEBD group III animals (GPIII) with 100 mg.kg^{-1} of **AKKINI CHOORANAM**, group IV animals (GPIV) with 200 mg.kg^{-1} of **AKKINI CHOORANAM**, and group V animals (GPV) with 400 mg.kg^{-1}**AKKINI CHOORANAM** The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01 *P<0.05.

Effect of AKKINI CHOORANAM on haematological parameters in rats

The effects of **AKKINI CHOORANAM**were observed for its effect onhaematological parameterson the experimental rats. From the study it was evident that, a significant increase (p<0.01) were observed in the haemoglobin contents and RBC count in the group treated with 200 mg.kg^{-1} body weight of **AKKINI CHOORANAM**and a significant decrease of the parameters occurred in the group treated with 400 mg.kg^{-1} b.w.t compared with the control. There was no significant change in the calcium level in all the treated animals compared to the control.

Treatment	Haemoglobin (mg.dl^{-1})	RBC (10^6 /mm^3)	WBC (10^6 /mm^3)	Calcium (mg.dl^{-1})
Control	13.52±1.28	9.25± 0.93	11.51± 0.90	9.42 ±0.60
AKKINI CHOORANAM @ 50 mg.kg^{-1}	14.33±1.35*	9.38±1.05*	9.34± 0.82*	9.24 ±0.38
AKKINI CHOORANAM @ 100 mg.kg^{-1}	14.21±1.84*	9.47±1.20*	8.34± 0.28*	9.24 ±0.45
AKKINI CHOORANAM@ 200 mg.kg^{-1}	13.26±1.25*	8.36± 0.85*	11.51± 0.83*	9.58 ±0.56
AKKINI CHOORANAM@ 400 mg.kg^{-1}	13.24±1.23*	8.48± 0.92*	10.84±0.75*	9.66 ±0.64

Table.6.The effect of **AKKINI CHOORANAM** on haematological parameters such as HB, Calcium, RBC and WBC in rats. A study on the effect of **AKKINI CHOORANAM** on haematological parameters such as Hb, RBC, WBC, Calcium in rats was tested. where, group I animals (GPI) treated with normal saline (5 $ml.kg^{1}$), group II animals (GPII) with 50 $mg.kg^{-1}$ of **AKKINI CHOORANAM**, group III animals (GPIII) with 100 $mg.kg^{-1}$ of **AKKINI CHOORANAM**, group IV animals (GPIV) with 200 $mg.kg^{-1}$ of **AKKINI CHOORANAM**, and group V animals (GPV) with 400 $mg.kg^{-1}$**AKKINI CHOORANAM**. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV and V. The statistical analysis was carried out using one way ANOVA method, where *P<0.05.

Discussion

The evaluation of sub-chronic and chronic dosing in experimental animals may be more relevant in determining the overall toxicity of the plant preparation. The highest overall concordance of toxicity in animals in comparison with humans is with hematological, gastrointestinal, and cardiovascular adverse effects whiles certain adverse effects in humans, especially hypersensitivity and idiosyncratic reactions, are poorly correlated with toxicity observed in animals (Olson, *et al*., 2000).(7)

In the present study, where the acute toxicity study of **AKKINI CHOORANAM** was carried out as per OECD-423 guidelines,no mortality was observed in both the animals of control group as well as animals treated with a maximum dose of 2000 $mg.kg^{-1}$. Hence, $1/10^{th}$ of 2000 $mg.kg^{-1}$ i.e. 200 $mg.kg^{-1}$ of dose was selected as a minimum dose for sub-acutetoxicity study (Abu TahaNael, *et al.,* 2008).(8)

The results of sub-acute toxicity study shows that there was no significant change in animal behaviour due to the absence of toxicity. The animals treated with **AKKINI CHOORANAM** showed normal growth pattern and body weight compared with control rats treated with normal saline. So the changes in body weight can be used as an indicator of adverse effects of drugs and chemicals (Tofovic and Jackson, 1999; Raza, *et al.*, 2002; Teo, 2002).(9,10,11)

The changes in enzymes like ALP, AST and ALT levels show liver impairment, due to toxicity (Hayes, 1989).(12) Serum cholesterol and proteins mainly regulated via synthesis in the liver and increase or decrease in serum concentrations of constituents suggest liver toxicity. The results of the present study were assessed after 28 days of administration of **AKKINI CHOORANAM**, and it was found that **AKKINI CHOORANAM** at all concentrations do not produce liver damage.

There was a slight decrease in plasma glucose level, when higher doses of **AKKINI CHOORANAM** (400 $mg.kg^{-1}$) were administered in the treated rats..

Analysis of blood parameters is likely to risk evaluation as the change in hematological system has a higher predictive value for human toxicity, when data are

translated from animal studies (Olson, *et al*., 2000).(7) After 28 days of treatment, there were no significant changes in the haematological parameters between control and treated groups. No significant changes in the levels of WBC, RBC were observed between control and test groups following repeated administration of **AKKINI CHOORANAM**. Interestingly, significant increase in the levels of hemoglobin was found in treatment with **AKKINI CHOORANAM** with a higher dose of 400 $mg.kg^{-1}$. The possible reason could be that one of the constituents **AKKINI CHOORANAM** may increase absorption of iron.

The overall results suggest that **AKKINI CHOORANAM** are non toxic to the haaematopoietic and leucopoietic system. The haematopoietic and leucopoietic systems are the most sensitive targets for toxic compounds and an important index of physiological and pathological status in man and animal (Adeneye, *et al*., 2006).(13) Therefore, it is possible to assume that the **AKKINI CHOORANAM**is non haematotoxic.

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PALAYAMKOTTAI, TIRUNELVELI

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A PROSPECTIVE, OPEN LABELLED, RANDOMIZED PHASE – II CLINICAL TRIAL ON **VALI AZHAL KEEL VAYU** (RHEUMATOID ARTHRITIS) WITH EVALUATION OF THE TRIAL DRUG "**AKKINI CHOORANAM**"

FORM - I

SCREENING AND SELECTION PROFORMA

01. Name of the subject :

02. S. No. of the Subject :

03. OP No : 04. IP No :

05. Date of Admisión :

06. Date of Discharge :

07. Address :

08. Contact Number :

09. Date of Birth : 10. Age (in yrs):

11. Code No (of clinical trial):

12. Gender : Male 1 Female 2

13. CRITERA OF INCLUSON Yes 1 No 2

01	Age between 18 and 50 years								
	18 - 20	1	21 - 30	2	31 - 40	3	41 - 50	4	
02	Both Sex		Male						
			Female						
03	Symmetrical joint involvement								

04	Arthritis of 3 or more joints	
05	Florid Morning stiffness and swelling that lasts for few hours or days	
06	Pain, Joint swelling especially in the inter phalangeal joint	
07	Polyarthritis	
08	Patients who are willing for admission and stay in IPD for minimum 20-30 days or willing to attend OPD	
09	Patient willing to sign the informed Patient who are willing to undergo radiological investigation and give blood and urine samples for laboratory investigation.	
10	Patient willing to sign the informed consent stating than he/she will consciously stick to the treatment during 30 days but can OPD out of the trial of his/her own conscious discretion.	
11	Pain criteria score should be greater than or equal to 6	
12	DISEASE ACTIVITY PAIN SCORE OF 28 JOINTS' should be greater than or equal to 3.2.	

14. CRTERIA FOR EXCLUSION

Yes	1	No	2

01	Age below 18 and above 50	
02	Systemic Hypertension	
03	Diabetes mellitus	
04	Chronic alcoholics, Chronic smokers	
05	Pregnancy and lactating Mothers	
06	Extra pulmonary Tuberculosis	
07	Psoriatic arthritis	
08	Gouty arthritis	
09	Chronic kidney disease	

A patient is eligible for admission

- If "Yes" or "No" : ☐
- If admitted:
  - ✓ Sr. No. of the Subject: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐
  - ✓ No. of packets issued : ☐ 4.1 grm / bd with Ghee or Sugar for 2 days.

Date : ☐☐ ☐☐ ☐☐☐☐ _____

Place : _____ Signature of the Investigator

_____ _____

Signature of guide Signature of Supervisor

Date : ☐☐ ☐☐ ☐☐☐☐ Date : ☐☐ ☐☐ ☐☐☐☐

Place : _____ Place : _____

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FORM – I A

HISTORY PROFOMA ON ENROLMENT

01. Name of the subject :

02. Sr. No. of the Subject :

03. OP No : 04. IP No :

05. Date of Admisión :

06. Date of Discharge :

07. Address :

08. Contact Number :

09. Date of Birth : 10. Age (in yrs):

11. Code No (of clinical trial) :

12. Gender : Male 1 Female 2

13. Educational status:

Illiterate	1	High school	5
Read and write	2	College	6
Primary	3	Others (specify)	7
Middle school	4	INA	8

14. Occupation

Desk work	1
Field work	2
Field work with physical labour	3
Field work with intellectual	4

Indicate Nature of work _____

13. Religion

Hindu	1	Christian	4
Muslim	2	Parsi	5
Sikh	3		

14. Total Family members: ☐ Children I. Male II. Female

12. Income per capita per month (in Rs): ☐

13. Chief complaint with duration (if any) in

Yes	1	No	2

No	Chief complaint	1/2	Duration days
01	Severe pain and swelling in small joints		
02	Symmetrical joint involvement		
03	Morning stiffness		
04	Anorexia		
05	Rheumatoid nodules		
06	Depression		
07	Others		

If Yes specify : _____

14. PERSONAL HISTORY

1. Diet

Veg	1	Non-veg	2	Mixed	3

2. Presence of anxiety

Yes	1	No	2

3. Constipation

Yes	1	No	2

4. Addiction

Yes	1	No	2

15. PERSONAL HABITS

I. Smoking

If yes specify: (a) Quantity packs :

(b) Total Duration in year's :

II. Tobacco

If yes specify: (a) Quantity :

(b) Total Duration in years :

III. Alcohol

If yes specify: (a) Quantity (in ml/day) :

(b) Total Duration in years :

IV. Any other (specify) : _____

16. Drug history: Had the patient been treated before with allopathy drug?

17. MARITAL STATUS :

Married	1	Unmarried	2

18. FAMILY HISTORY :

Whether this problem runs in family?

Yes	1	No	2

If yes, mention the relationship of affected person (s) : _____

Normal	1	Abnormal	2

19. BOWEL HABITS & MICTURITION:

✓ History of habitual constipation	Yes	1	No	2
✓ History of frequent diarrhoea	Yes	1	No	2
✓ History of frequent dysuria	Yes	1	No	2

20. PSYCHOLOGICAL STATE:

Normal	1	Anxiety	2	Depression	3

21. PRAKRITI :

Vatham	1	Kapham	3	Vatha-kapham	5	Sannipatham	7
Pitham	2	Vatha-pitham	4	Pitha-Kapham	6		

22. PHYSICAL EXAMINATION

1. Height : ☐ cm
2. Weight : ☐ kg
3. Pulse Rate : ☐ per min
4. Heart Rate : ☐ per min
5. Blood Pressure (in sitting position)
   - ✓ Systolic : ☐ mm Hg
   - ✓ Diastolic : ☐ mm Hg
6. Body temperature : ☐ ^{o}F
7. Respiration rate : ☐ per min
8. Signs of dehydration and oedema, if any: _____

23. SYSTEMC EXAMINATION

Normal	1	Abnormal	2

1. Cardio Vascular System

If abnormal, details : _____

2. Central Nervous System

If abnormal, details : _____

3. Digestive system

If abnormal, details : _____

4. Respiratory system

If abnormal, details : _____

5. Locomotor System

If abnormal, details : _____

6. Endocrine System

If abnormal, details : _____

7. Genito-urinary system

If abnormal, details : _____

Date : ☐☐ ☐☐ ☐☐☐☐

Place : _____

_____

Signature of the Investigator

_____

Signature of guide

Date : ☐☐ ☐☐ ☐☐☐☐

Place : _____

_____

Signature of Supervisor

Date :

Place : _____

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FORM – II A

CLINICAL ASSESSMENT ON ENROLMENT AND ON VISITS

01. Name of the subject :

02. S. No. of the Subject :

03. OP No : 04. IP No :

05. Date of Admisión :

06. Date of Discharge :

07. Address :

08. Contact Number :

09. Date of Birth : 10. Age (in yrs):

11. Code No (of clinical trial) :

12. Gender : Male 1 Female 2

SIDDHA SYSTEM OF EXAMINATION

1. ENVAGAI THERVU: (Eight-Fold Examination)

I. NAADI (Pulse perception)

Naadi	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Vazhi							
Azhal							
Iyyam							
VazhiAzhal							
VazhiIyyam							
AzhalVazhi							
AzhalIyyam							
IyyaVazhi							
IyyaAzhal							

II. NAA (Tongue)

Colour		Taste		Mozhi		Salaiva, Nurai Edai Volume		Consistency		Coating, Dryness Glossitis, Baldness Fissure, Diarrhoea Manam, Enjal	
Dark	1	Sweet	1	High	1	Normal / Nil	1	Solid	1	Present	1
Yellow	2	Bitter	2	Medium	2	Increased	2	Watery	2	Absent	2
Red	3	Sour	3	Low	3	Decreased	3	Semisolid	3		
Pale	4	Pungent	4								
Tinted	5	None	5								

Naa	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Colour							
Taste							
Coating							
Fissure							
Saliva							
Dryness							
Glossitis							
Baldness							

III. NIRAM (Complexion)

Niram	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Dark							
Yellow							
Tinted							
Pale							

IV. MOZHI (Voice)

Mozhi	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Medium							
High							
Low							
Pitched							

V. **VIZHI (Eyes) (Lower Palpebral Conjunctiva)**

Niram	**0stDay**	**05thDay**	**10th Day**	**15th Day**	**20th Day**	**25th Day**	**30thDay**
Dark							
Yellow							
Red							
Pale							

VI. **MALAM (Bowel habits / Stools)**

Naa	**0stDay**	**05thDay**	**10th Day**	**15th Day**	**20th Day**	**25th Day**	**30thDay**
Colour							
Consistency							
Stool bulk							
Constipation							
Diarrhea							

VII. URINE EXAMINATION

Neerkuri	**0stDay**	**05thDay**	**10th Day**	**15th Day**	**20th Day**	**25th Day**	**30thDay**
Niram (Colour)							
Manam (Odour)							
Nurai (Froth)							
Edai (weight/10 ml)							
Enjal (Deposits)							
Volume							

Neikuri	**0stDay**	**05thDay**	**10th Day**	**15th Day**	**20th Day**	**25th Day**	**30thDay**
Serpentine fashion							
Annular/Ringed fashion							
Pearl beaded Fashion							
Mixed fashion							
Other fashion							

VIII. SPARISAM (Palpatory perception)

Sparisam	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Warmth/heat							
Cold							
Sweat							

2. THEGI (type of body constitution)

Vatham predominant	1
Kabam predominant	2
Pitham predominant	3
Thondhaudal	4

3. NILAM (land where patient lived most)

Kurinji (Hilly terrain)	1
Mullai (Forest range)	2
Marutham (Plains)	3
Neithal (Coastal belt)	4
Palai (Arid regions)	5

4. KAALAM

Kaarkalam	1	Pinpanikalam	4
Koothirkalam	2	Ilavenil	5
Munpanikalam	3	Muthuvenil	6

5. GUNAM

Sathuvam	1
Rasatham	2
Thamasam	3

6. IMPORIGAL (Sensory Organs)

Organs	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Mei (Skin)							
Vai (Buccal Cavity)							
Kann (Eye)							
Sevi (Ear)							
Mooku (Nose)							

7. KANMENDRIYAM (MOTOR FUNCTIONS)

Kanmendriyam	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Kai(Upperlimb)							
Kaal(lowerlimbs)							
Vai(buccalcavity)							
Eruvaai (Excretory organs)							
Karuvaai (Reproductive organs)							

8. KOSANGAL (Sheath)

Kosangal	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Annamaya Kosam							
Pranamaya Kosam							
Manomaya Kosam							
Vignanamaya Kosam							
Ananthamaya Kosam							

9. MUKKUTRAM (AFFECTION OF THREE HUMORS)

A) VATHAM

Vatham	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Praanan							
Abaanan							
Viyaanan							
Udhaanan							
Samanan							
Naagan							
Koorman							
Kirukaran							
Devathathan							
Dhananjeyan							

B) PITHTHAM

Piththam	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Analapitham							
Ranjagam							
Saathagam							
Praasagam							
Aalosagam							

C) KAPHAM

Kapham	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Avalambagam							
Kilethagam							
Pothagam							
Tharpagam							
Santhigam							

10. SEVEN DHATHUS (7 SOMATIC COMPONENTS)

Dhathus	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Saaram (Chyme)							
Senneer (Blood)							
Oon (Muscle)							
Kozhuppu (Fat)							
Enbu (Bones)							
Moolai (Bone Marrow)							
Sukkilam / Suronitham							

11. SYSTEMIC EXAMINATION

Systems	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Locomotor system							
Cardiovascular system							
Respiratory system							
Gastro intestinal system							
Central nervous system							
Genito-urinary system							
Endocrine system							

12. GENERAL EXAMINATION

	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Height (cms)							
Weight (kg)							
Temperature (F^0)							
Pulse rate (per min)							
Heart rate (per min)							
Respiratory rate (per min)							
Pallor							
Jaundice							
Cyanosis							
Lymph adenopathy							
Pedal edema							
Clubbing							
Jugular vein pulsation							

13. EXAMINATION OF THE INTERNAL ORGANS

Organs	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Brain							
Lungs							
Liver							
Pancreas							
Kidney							
Urinary Bladder							
Heart							
Stomach							
Gall Bladder							
Intestines							
Uterus							
Rectum							

14. CLINICAL SYMPTOMS

Complaints	0st Day	05th Day	10th Day	15th Day	20th Day	25th Day	30th Day
Pain and swelling in small joints							
Symmetrical joint involvement							
Morning stiffness							
Anorexia							
Rheumatoid nodules							
Depression							

15. CLINICAL SYMPTOMS:

CRITERION JOINTS AFFECTED	NORMAL SCORE	PATIENT'S SCORE
1 Large joint	0	
2-10 Large joints	1	
1-3 Small joints	2	
4-10 Small joints	5	
SEROLOGY		
Negative RF and Accp	0	
Low Positive RF or Accp	2	
High Positive RF or Accp	3	
DURATION OF SYMPTOMS		
<6 weeks	0	
>6 weeks	1	
ACUTE PHASE REACTANTS		
Normal CRP and ESR	0	
Abnormal CRP or ESR	1	

Patient with a score greater than or equal 6 are considered to have definite Rheumatiod Arthritis.

16. CLINICAL ASSESSMENTOF AFFECTED JOINT

INSPECTION:

Inspection	Present	Absent
Symmetry		
Swelling		
Joint deformity		
Muscle wasting		

If present, _____

Range of motion: Affected / Not affected

PALPATION:

Inspection	Present	Absent
Symmetry		
Warmth		
Tenderness		
Crepitation		

If present, _____

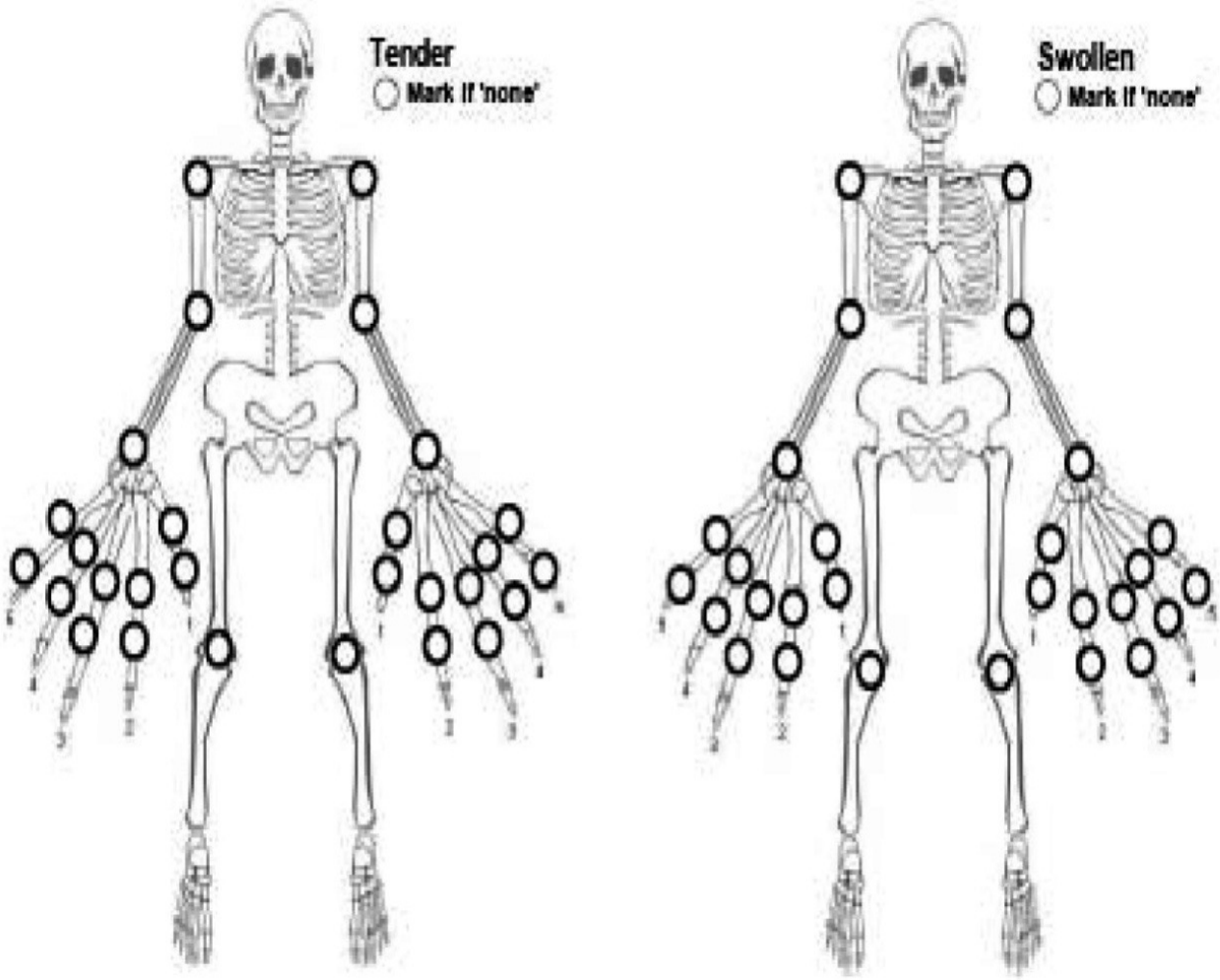
MOVEMENTS:

1. Restriction of movements : Full ☐ Partial ☐ NO ☐
2. Movements:

Movements	Pain		Muscular spasm		Range of movement	
	Yes	No	Yes	No	Normal	Reduced
i). Flexion						
ii). Extension						
iii). Abduction						
iv). Adduction						
v). Circumduction						
vi). Rotation						
vii). Lateral rotation (External)						
viii). Medical rotation (Internal)						
ix). Others						

DISEASE ACTIVITY SCORE OF 28 JOINTS (DAS 28)

VERY WELL |_|_|_|_|_|_|_|_|_|_| VERY POOR



VAS (0-100) 28TJC 28SJC ESR

DAS 28 = 0.56 × $\sqrt{(28TJC)}$ + 0.28 × $\sqrt{28\ (SJC)}$ + 0.70 × In (ESR) + 0.14 × VAS

DAS 28 ☐

Interpretation:

Low disease activity	:	2.6<DAS28≤3.2
Moderate disease activity	:	3.2<DAS28≤5.1
High disease activity	:	DAS28>5.1

Before treatment	After treatment

Date : ☐☐ ☐☐ ☐☐☐☐

Place : _____

_____

Signature of the Investigator

_____

Signature of guide

Date : ☐☐ ☐☐ ☐☐☐☐

Place : _____

_____

Signature of Supervisor

Date : ☐☐ ☐☐ ☐☐☐☐

Place : _____

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL

PALAYAMKOTTAI, TIRUNELVELI

DEPARTMENT OF POTHU MARUTHUVAM

A PROSPECTIVE, OPEN LABELLED, RANDOMIZED PHASE – II CLINICAL TRIAL ON **VALI AZHAL KEEL VAYU** (RHEUMATOID ARTHRITIS) WITH EVALUATION OF THE TRIAL DRUG "**AKKINI CHOORANAM**"

FORM – II B

BEFORE TREATMENT & FORTIGHTLY DURING TREATMENT

01. Name of the subject :

02. S. No. of the Subject :

03. OP No : 04. IP No :

05. Date of Admisión :

06. Address :

07. Contact Number :

08. Date of Birth : 09. Age (in yrs):

10. Code No (of clinical trial) :

11. Gender : Male 1 Female 2

12. Date of Assessment :

13. Chief complaint with duration (if any) in Absent 1 Present 2

No	Chief complaint	1or 2	Duration
01	Severe pain and swelling in small joints		
02	Symmetrical joint involvement		
03	Morning stiffness		
04	Anorexia		
05	Rheumatoid nodules		
06	Depression		
07	Severe pain and swelling in small joints		

If Yes specify : _____

14. Physiological Assessment

- ✓ Weight (in kgs) :
- ✓ Blood Pressure (in sitting position) Systolic (mm Hg) :
  Diastolic (mm Hg) :

Date :
Place : _____

_____
Signature of the Investigator

_____
Signature of guide
Date :
Place : _____

_____
Signature of Supervisor
Date :
Place : _____

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL

PALAYAMKOTTAI, TIRUNELVELI

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A PROSPECTIVE, OPEN LABELLED, RANDOMIZED PHASE – II CLINICA

TRIAL ON VALI AZHAL KEEL VAYU (RHEUMATOID ARTHRITIS) WITH

EVALUATION OF THE TRIAL DRUG "AKKINI CHOORANAM"

FORM - III

LABORATORY PARAMETERS - CHART

01. Name of the subject :

02. Sr. No. of the Subject :

03. OP No : 04. IP No :

05. Date of Admisión :

06. Date of Discharge :

07. Address :

08. Contact Number :

09. Date of Birth : 10. Age (in yrs):

11. Code No (of clinical trial) :

12. Gender : Male 1 Female 2

13. Bed No :

❖ **LAB INVESTIGATION CHART**

Blood Investigation		**Normal Values**	**Before TMT (With Date)**	**In Between (With Date)**		**After TM (With Dat**
Hb (gms%)		M : 12 - 15 W : 11.5 - 14				
T.RBC(milli/cu.mm)		M : 4.0 - 5.5 W : 3.5 - 4.5				
ESR (mm /hr)	1/2 hr	————				
	1 hr	M : 6 - 12 W : 7 - 18				

T.WBC (cells /cu.mm)		4000 - 10000				
Differential Count (%)	Polymorphs	40 - 75				
	Lymphocytes	20 - 40				
	Monocytes	02 - 10				
	Esionophils	01 - 06				
	Basophils	00 - 01				
Platelets ; (lak/ cubic mm)		1,50000-500000				
Blood glucose (mg/dl)	Fasting	< 100				
	PP	< 140				
	Random	< 120				
Serum Cholesterol		< 200				
Urea						
Creatinine						
Uric Acid						

Urine investigation	**Before Treatment (with Date)**	**InBetween (WithDate)**		**After Treatment (With Date)**
Neerkuri				
Neikuri				
Niram				
Manam				
Nurai				
Edai				
Enjal				

Albumin				
Sugar (F)				
Sugar (PP)				
Sugar (R)				
Deposits				

❖ **SPECIFIC INVESTIGATIONS**

	Before treatment	**After treatment**
CRP		
RA Factor		
ASO Titer		

❖ **RADIOLOGICAL INVESTIGATIONS**

X- ray of affected joints (AP and Lateral view)

Date : ☐☐ ☐☐ ☐☐☐☐ _____

Place : _____ Signature of the Investigator

_____ _____

Signature of guide Signature of Supervisor

Date : ☐☐ ☐☐ ☐☐☐☐ Date :

Place : _____ Place : _____

அரசினர் சித்த மருத்துவக் கல்லூரி மற்றும் மருத்துவமனை

பாளையங்கோட்டை

பட்டமேற்படிப்பு பொதுமருத்துவத்துறை

வளிஅழல்கீல்வாயுவிற்கு (RHEUMATOID ARTHRITIS)

அக்கினிச்சூரணத்தின் பரிகரிப்புத் திறனைக் கண்டறியும்

மருத்துவஆய்வு ஒப்புதல் படிவம்

ஆய்வாளரின் சான்றிதழ்

நான் இந்தஆய்வுகுறித்தஅனைத்துவிபரங்களையும் நோயாளிக்குப் புரியும் வகையில் எடுத்துரைத்தேன் என உறுதியளிக்கிறேன்.

பெயர் : _____

கையொப்பம் : _____

தேதி : _____

இடம் : _____

நோயாளியின் ஒப்புதல்

என்னிடம் இந்தமருத்துவஆய்வின் காரணத்தையும், மருந்தின் தன்மை மற்றும் மருத்துவ வழிமுறையைப்பற்றியும் தொடர்ந்து எனது உடல் இயக்கத்தை கண்காணிக்கவும் அதனைப் பாதுகாக்கவும் பயன்படும் மருத்துவ ஆய்வுகூட பரிசோதனைகள் பற்றி திருப்தி அளிக்கும் வகையில் ஆய்வு மருத்துவரால் விளக்கிக் கூறப்பட்டது.

நான் இந்த மருத்துவ ஆய்வின்போது காரணம் எதுவும் கூறாமல் எப்பொழுது வேண்டுமானாலும் இந்த ஆய்விலிருந்து என்னை விடுவித்துக் கொள்ளும் உரிமையை தெரிந்திருக்கிறேன்.

நான் என்னுடைய சுதந்திரமாக தேர்வு செய்யும் உரிமையைக் கொண்டு வளிஅழல்கீல்வாயு (RHEUMATOID ARTHRITIS) நோய்க்கு மருந்தாக அக்கினி சூரணத்தின் பரிகரிப்புத் திறனைக் கண்டறியும் மருத்துவ ஆய்விற்கு என்னை உட்படுத்த ஒப்புதல் அளிக்கிறேன்.

பெயர் : _____ சாட்சியின் பெயர் : _____

கையொப்பம் : _____ சாட்சியின் கையொப்பம் : _____

தேதி : _____ உறவுமுறை : _____

இடம் : _____ தேதி : _____ இடம் : _____

தேதி : _____ _____

இடம் : _____ மேற்பார்வையாளர் கையொப்பம்

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL

PALAYAMKOTTAI, TIRUNELVELI

DEPARTMENT OF POTHU MARUTHUVAM

A PROSPECTIVE, OPEN LABELLED, RANDOMIZED PHASE – II CLINICA

TRIAL ON VALI AZHAL KEEL VAYU (RHEUMATOID ARTHRITIS) WITH

EVALUATION OF THE TRIAL DRUG "AKKINI CHOORANAM"

FORM – IV A

CONSENT FORM

CERTIFICATE BY INVESTIGATOR

I certify that I have disclosed all details about the study in the terms easily underst the patient.

Name of Investigator : **DR.P.PRIYANGA**

Date : _____ Signature of the Investigator : _____

CONSENT BY SUBJECT

I have been informed to my satisfaction, by the attending physician, the purpose clinical trial, and the nature of drug treatment and follow-up including the laboratory investi to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of t without having to give the reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included

As a subject in An Open Labeled Randomized Clinical Trial of Poly Herbal Formula **"AKKINI CHOORANAM"** for **VALI AZHAL KEEL VAYU (RHEUMATOID ARTH**

❖ Name of the Subject : _____

Date : _____ Signature or Thumb impression : _____

❖ Name of witness : _____

Date : _____ Signature or Thumb impression : _____

Relationship : _____

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TRIAL ON VALI AZHAL KEEL VAYU (RHEUMATOID ARTHRITIS) WITH

EVALUATION OF THE TRIAL DRUG "AKKINI CHOORANAM"

FORM – IV B

WITHDRAWAL FORM

01. Name of the subject :

02. S. No. of the Subject :

03. OP No : 04. IP No :

05. Date of Admisión :

06. Address :

07. Contact Number :

08. Date of Birth : 09. Age (in yrs):

10. Code No (of clinical trial) :

11. Gender : Male 1 Female 2

12. Date of trial commencement :

13. Date of withdrawal from trial :

Reasons for withdrawal

Yes 1 No 2

Long absence at reporting	
Irregular treatment	
Shift of locality	
Increase in severity of symptoms	
Development of severe adverse drug reactions	

Date :

Place : _____

_____

Signature of the Investigator

_____

Signature of guide

Date :

Place : _____

_____

Signature of Supervisor

Date :

Place : _____

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL

PALAYAMKOTTAI, TIRUNELVELI

DEPARTMENT OF POTHU MARUTHUVAM

A PROSPECTIVE, OPEN LABELLED,RANDOMIZED PHASE – II CLINICA

TRIAL ON VALI AZHAL KEEL VAYU (RHEUMATOID ARTHRITIS) WITH

EVALUATION OF THE TRIAL DRUG "AKKINI CHOORANAM"

FORM – IV C

PATENT INFORMATION SHEET

- Name of the Principal Investigator : **DR.P.PRIYANGA**
- Name of the Institution : Government Siddha Medical College & Hospit
  Palayamkottai, Tirunelveli
  Tamil Nadu

- I, Dr.P.Priyanga studying M.D (Siddha) in Government Siddha Medical Co Palayamkottai. The disease called **VALI AZHAL KEEL VAYU (RHEUMAT ARTHRITIS).** It is mainly caused due to vitiated Vatha and Pitha humours.

- This condition is being treated in GSMC & H with many siddha formulations. As a pa M.D (s) research programme and developing new efficacious medicine, I propose to the **AKKINI CHOORNAM** formulation for treating the condition. This formulatio been mentioned in siddha literature and empirical evidence with contemporary too required for documentation. You can receive medicines free of cost. The duratio treatment period is 30 days. You have to visit GSMC & H every 2 days and collect for **2 days.** The diagnosis tests will be carried out free of cost. We will assess the effe treatment after completion of **30 days** of treatment using clinical and lab parameters.

- The trial drug is prepared at the Gunapadam lab of government siddha medical colle hospital, palayamkottai, under the direct supervision of teaching faculties of Maruth and Gunapadam Dept.

- Patients are advised to do exercise, reduce the intake of salt. Patients are advised to tamarind, betel chewing, tobacco, alcohol and smoking.

Details of the trial drug

- ✓ Trial Medicine : Akkini Chooranam
- ✓ Dosage : 4.1 gram – twice a day
- ✓ Adjuvant : Ghee or Sugar
- ✓ Duration : 30 days

- ❖ In this regard, I need to ask you few questions. We will maintain confidentiality of comments and data obtained from you. There will be no risk of disclosing your identity no physical, psychological or professional risk is involved by taking part in this study.
- ❖ Taking part in this study is voluntary. No compensation will be paid to you for taking p this study. You can choose not to answer any specific question. There is no specific be for you if you take part in the study, but you will be under our clinical monitoring specific attention will be given for your health. Taking part in the study may be of bene the community, as it may help us to develop medicine for VALI AZHAL KEEL V (RHEUMATOID ARTHRITIS). In case of any adverse symptoms during the treatme few patients during the treatment, shall be reported to PIs and care will be taken in GSM H for relief. You can withdraw from the study at the midst of treatment period, if yo not interested to continue and you will receive our usual treatment without condition.
- ❖ The information we will collect in this study, will remain between you and the prin investigator. We will ask you a few questions through questionnaire. We will not write name on different forms which sent to different investigating / analysis sections and we use a code instead given by the principal investigator. Only the principal investigator know the key to this code which will be kept in safe custody. If you agree to be a partic in this study, you will be screened as per the study protocol.
- ❖ If you wish to find out more about this study before taking part, you can ask me a questions you want or contact Dr.P.PRIYANGA, PG scholar cum principal investigat this study, attached to the Government Siddha Medical College & Hospital, Palayamk (Mobile Phone No : 8489023383).

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A PROSPECTIVE, OPEN LABELLED,RANDOMIZED PHASE – II CLINICAL TRIAL ON **VALI AZHAL KEEL VAYU** (RHEUMATOID ARTHRITIS) WITH EVALUATION OF THE TRIAL DRUG "**AKKINI CHOORANAM**"

FORM – IV D

ADVERSE DRUG REACTION FORM

01. Name of the subject :

02. S. No. of the Subject :

03. OP No : 04. IP No :

05. Date of Admisión :

06. Address :

07. Contact Number :

08. Date of Birth : 09. Age (in yrs):

10. Code No (of clinical trial) :

11. Gender : Male 1 Female 2

12. Date of trial commencement :

13. Date of withdrawal from trial :

14. Description of adverse reaction : _____

_____

Date :

Place : _____

_____

Signature of the Investigator

_____

Signature of guide

Date :

Place : _____

_____

Signature of Supervisor

Date :

Place : _____

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL

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A PROSPECTIVE, OPEN LABELLED,RANDOMIZED PHASE – II CLINICAL TRIAL ON VALI AZHAL KEEL VAYU (RHEUMATOID ARTHRITIS) WITH EVALUATION OF THE TRIAL DRUG "AKKINI CHOORANAM"

FORM – IV E

DRUG COMPLIANCE FORM

01. Name of the subject :
02. S. No. of the Subject :
03. OP No : 04. IP No :
05. Date of Admisión :
06. Date of Discharge
07. Address :
08. Contact Number :
09. Date of Birth : 10. Age (in yrs):
11. Code No (of clinical trial) :
12. Gender : Male 1 Female 2
08. Bed No :
10. Name of the Drug : AKKINI CHOORANAM
Drugs issued :
Drugs returned date :

S.No	Date	Drug Taken Time		
		Morning / Time	Noon / Time	Night / Time
Day 01				
Day 02				
Day 03				
Day 04				
Day 05				
Day 06				

Day 07				
Day 08				
Day 09				
Day 10				
Day 11				
Day 12				
Day 13				
Day 14				
Day 15				
Day 16				
Day 17				
Day 18				
Day 19				
Day 20				
Day 21				
Day 22				
Day 23				
Day 24				
Day 25				
Day 26				
Day 27				
Day 28				
Day 29				
Day 30				

Date : ☐☐ ☐☐ ☐☐☐☐

Place : _____

_____

Signature of the Investigator

_____

Signature of guide

Date : ☐☐ ☐☐ ☐☐☐☐

Place : _____

_____

Signature of Supervisor

Date :

Place : _____